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EYE CHANGES IN GENERAL MEDICINE.[1]

By BRIAN MOORE, M.B., B.S. (Adelaide),
Adelaide.

OPHTHALMOLOGY is a subject given to rather verbose literature. It is extraordinary what can be written about so small a portion of the human anatomy, the reason being of course that our eyes are paired organs of special sense and vision is an all-important faculty, and eye changes are often of the greatest importance to the neurologist and physician. In writing this paper, I have been faced with the difficulty mainly of knowing what to leave out rather than what to put in. The field of neurology is an attractive one, but after I had written half of a paper dealing with its connexion with eye changes, I found myself being led further and further along involved paths, which would be of interest to only a few people. I have therefore tried to attack the subject of this paper from a different angle, really from the point of view of the general practitioner, who is often called upon to advise his patients in connexion with eye signs and symptoms, and who may be glad to refresh his memory of how to diagnose certain eye diseases and what advice to give as a result. I hope that what I am going to say will not be too elementary.

My own recollections of what I knew about ophthalmology when in general practice and the advice I occasionally gave, now that I know a little more do not leave me with very satisfactory thoughts to dwell upon. Possibly I was more ignorant than most, but if there are a few here who may be helped by what I am going to say, I hope the more enlightened amongst you will bear with me for their sake.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 25, 1932.

Method of Examination.

I think it reasonable to start with some mention of a method of examination, without which it is impossible to arrive at a correct interpretation of the eye changes under our notice.

It is much better to have the same routine for every case. To go straight to the affection complained of and examine this alone, will lead to other and more important changes being missed more often than not. For example, a small corneal ulcer may resist treatment and remain irritable, if one misses the fact that there is a displaced lash growing inwards and not allowing it to heal.

I would suggest that the examination commence with a record of the distance vision with Snellen's test types. If the patient wears glasses, the vision should be taken through them. It would not be of any advantage to know that a highly ametropic person cannot read $^{6}/_{60}$, whereas with his glasses he may read $^{6}/_{6}$.

Following the estimation of distance vision, see if the patient can read Jaeger's type No. 1. People of presbyopic age will not be able to do this without suitable reading glasses, but if a young person, who can see $^{6}/_{6}$, cannot do so, it shows either very high hypermetropia or a paralysis of accommodation which may have followed an attack of diphtheria otherwise unrecognized.

An inspection of the patient in good daylight should follow. Note the general contour of the patient's face, particularly the eyebrows and orbits and the relative position of the lids with regard to the globes. The position of the eyes can be observed for exophthalmos or enophthalmos, these conditions being recognized by standing behind the seated patient and looking down from above while gently retracting the upper lid.

The action of the *levator palpebræ* and the frontalis muscle should be examined, and the action of the muscles of the globe by getting the patient to follow a finger held at about a metre away and moved into the nine principal positions.

A rough but helpful idea of the patient's field of vision can be obtained from each eye separately by moving a small white object, such as a piece of paper, halfway between the observer and the observed and noting if the patient's field corresponds fairly well with one's own eye. Small changes would not be sought for in this way, but a hemianopia would be easily recognized, and even the loss of field from a moderately advanced glaucoma or the temporal defect in pituitary tumour would be noticed, and also scotomata from retinal or chorioidal changes, such as a simple detachment or a chorioidal tumour.

Following this, the pupils should be examined as to size, shape and reactions to light and accommodation; then a more detailed examination of the lids and lashes conjunctiva and globe. One must not forget to evert both upper and lower lids and to examine the puncta and lachrymal sacs. Pressure over the latter may reveal the presence of a mucocele which is the real cause of some chronic irritation which otherwise would pass unnoticed.

To examine the cornea, anterior chamber and iris it is necessary to use a lamp, the light of which is focused by a lens of about 13 diopter strength on to the eye. This is an extremely simple thing to carry, and even if a darkroom is not avaliable, it will reveal changes which otherwise would not be seen. The light should be about one metre away from the patient, and the lens held at right angles to the rays of light, so that the focused light can be moved about into various positions of the eye under observation.

Corneal changes can be observed (better, of course, if a magnifying glass is used in conjunction) and any pathological change in the anterior chamber, pupil or iris looked for.

Following this, the eye is examined with an ophthalmoscope. This is better done in a darkened room, and I should like to enlarge a little on the use of this instrument. In these days every physician carries and uses one. They are of the electric self-illuminating type, and all have a device for introducing *plus* and *minus* lenses over the aperture through which the observation is made. These lenses, according to my observation, are not put to their full use. It should be more widely recognized that in every case the reflex should be obtained with the +20 lens. With this, by a slight movement of the observer's head, the anterior portion of the eye can be focused under great magnification, with the advantage of reflected light to view opacities or foreign bodies *et cetera,* in or on the cornea and precipitates on its posterior surface. By rotating the lens-race so that a lens of gradually diminishing convexity is used, the entire eye can be examined from cornea to retina for conditions in succession, such as pigment on the lens from old iritis, lens opacities and vitreous opacities.

Having arrived at the retina by the time sufficient convexity of lens has been removed, the examiner can go on to make his observations on the disk, retinal vessels *et cetera.* I consider it most important that this routine should be gone through in every fundus examination, as I have so often seen cases in which the physician has interpreted as fundus changes conditions in the media, such as lens or vitreous opacities, which cause a blurred image of the disk and retinal vessels.

I should like also to emphasize that no opinion of any important fundus condition should be given without an examination with a dilated pupil. I should never attempt to do it myself unless there were some reason which prevented its being carried out, and this must happen but seldom. In young children who are restless or cry, I think it necessary to use a general anæsthetic, if anything of importance hangs on discovering the fundus change. One drop each of a 2% solution of homatropine and cocaine is as good as anything else in young and middle-aged patients. In older people a 2% solution of cocaine is usually sufficient, and

may be safer, although if every case is followed up by instilling eserine, the dangers are negligible. To describe an examination is always more cumbersome than to carry it out. What I have taken some time to say may be easily done in a few minutes, and although it does not by any means comprise a complete ocular examination, it covers the ground necessary for the physician. I have purposely said nothing of an examination of the tension by the fingers, as few people have the necessary touch to place any reliance on it, unless they are continually doing eye examinations, and even then an exact estimation is very difficult. Any departure from normal observed during the examination would have to be followed up and gone into more thoroughly. Fundus examination is mentioned later in the paper. Following the order of the examination made, I should like to discuss a few of the conditions which the physician may meet and on which his advice may be sought.

Disorders of Vision.

In connexion with disorders of vision various symptoms may be complained of. If the complaint is of actually defective sight, an examination such as I described will give a very fair idea as to whether the defect is the result of a refractive error or of disease of the eye. To show more or less the complaints that people make whose eyes need correction, I tabulated one thousand consecutive new cases of my own, starting from the beginning of 1930, with the following results: 160 complained of eyes feeling tired, 180 complained of headaches, 102 complained of eyes feeling sore, 224 complained of defective vision, 334 were suffering from various medical and surgical eye conditions.

The result was of interest to me also in showing what a large proportion of patients in an ordinary ophthalmic practice were people who needed testing for glasses. It can be seen from these figures that it is in the neighbourhood of 67%. The number that actually were prescribed in this thousand was 618, as 48 were found on examination to have symptoms due to other causes.

I suppose of these symptoms the one which most closely concerns the physician, is headache. Not every headache by any means is connected with vision or eye strain. It has been estimated that 40% of all headaches and 75% of bilateral frontal headaches are caused by eye strain. In an ametropic person the ciliary muscles and also the ocular muscles themselves attempt to correct the ametropia and it is these efforts at correction which bring about the symptoms possibly, one supposes, by irritation of the third nerve nucleus.

The headache is almost invariably bilateral, and I would go so far as to say that unilateral supraorbital headache is practically always due to some other cause, frequently a frontal sinusitis or infection of the anterior ethmoidal cells. I should like to quote a case here which illustrates this.

I saw a patient, one with very high astigmatic correction, who after he had dropped his glasses developed an acute pain on the left side of his forehead. The left lens of his glasses had come out and been replaced. I found it had been put back twenty degrees away from its correct place, and his vision with it was only $^{6}/_{36}$. I rotated it to the correct position for him, whereupon he saw $^{6}/_{6}$, but I referred him at the same time to a nose and throat specialist, who found he had a frontal sinusitis. His pain did not abate one bit until the latter condition was dealt with. The development of his pain was a coincidence, and quite likely he would have noticed the blurred vision and come for its rectification only, if some other cause had not been operating.

Infections of the maxillary or sphenoidal sinuses very rarely give rise to symptoms of asthenopia. The position of the pain complained of in eye strain is more commonly supraorbital; less commonly the pain will be described as behind the eyes, while it may be referred to the occipital and, more rarely still, to the temporal region. The point I wish to stress is that the pain is almost invariably bilateral and that it is most commonly supraorbital.

The time at which the pain is complained of is important. Usually it follows and has a definite relationship to near work, but also in various refractive errors it may follow distant vision of a difficult kind, such as watching moving pictures or a cricket match. Sometimes the pain is described as appearing in the morning after the eyes have been used, but usually it starts during the performance of the work and increases till the work is put aside, when it disappears again. As a general rule it may be said that a headache which increases as the day goes on will most probably be due to eye strain, while one which is present on waking up and disappears during the morning is more likely to be due to an accessory nasal sinus infection.

Quite a number of patients who complain of eye strain have quite good distant vision. A young hypermetrope can read $^{6}/_{6}$ simply by exercising his powers of accommodation. It is the effort in excess of normal to do this which produces his symptoms. Naturally a slight or moderate diminution of vision down to $^{6}/_{9}$ or $^{6}/_{12}$ would confirm the view that a refractive error was present. These patients would probably be found to have astigmatic errors, but the patient with high refractive errors often complains only of his defective sight. Myopes rarely complain of headache. The errors of muscle balance or heterophorias which are responsible for many cases of eye strain, usually are accompanied by refractive errors. In my experience the discomfort from these conditions is largely ascribed to the eyes themselves.

To sum up, therefore, one must always consider eye strain as a possible factor in any case of headache. If the symptoms be concerned with slightly defective vision, the possibility is increased; while, if the headache is bilateral and frontal, associated with reading or with difficult distance vision, the possibility becomes almost a certainty.

It must be remembered that the symptoms of eye strain may manifest themselves in people without refractive errors whose work is mainly close, and especially if they are overtired and their general

tone is poor. These patients should be given a rest from duty and treatment on general lines.

Presbyopia.

Under the heading of disorders of vision I should like to discuss presbyopia, although this is not a pathological but a physiological condition. There seems to exist a certain doubt in some people's minds as to what actually happens to us when we find ourselves in the middle forties unable to look up a number in the telephone book with any certainty. In youth the powers of accommodation are large and in old age very small. The diminution goes on regularly and gradually all our lives. A boy of eight can normally exercise at least fourteen diopters of accommodation. That is to say, he can by using all his accommodation focus an object held seven centimetres away from his eyes, while the same person at forty-six has only a little over three diopters, which means by exercising all his powers he can see clearly at only about thirty centimetres. Obviously this effort cannot be kept up indefinitely, so that it is necessary to wear a *plus* lens to help his accommodation, the lens having to be increased in power at intervals of about three years, until at the age of fifty-five or so, provided his refraction does not change, he can settle down to wearing the same glasses without further alteration. Presbyopic people rarely suffer from headaches as a result of non-correction, though some do. Usually the symptoms of defective vision for near objects is what they describe, while a fairly common complaint is that their lids feel heavy and are difficult to open on waking.

Palpebral Muscles.

The changes from normal in the palpebral muscles which may meet the physician's eye are spasm and paralysis. Spasm of the orbicularis is easily recognized and, if due, as it usually is, to disease or irritation of the conjunctiva or globe, is removed by curing that condition. There is a type, however, which might be called an essential blepharospasm when no ocular change is present and yet the patient may find it at times quite impossible to open his eyes. These cases are extremely difficult to cure, and any special effort the patient makes to open his eyes increases the spasm. I saw one case last year in which the condition was combined with a refractive error and it was necessary to paralyse the orbicularis with "Novocain" and insert a lid retractor before the testing could be done. Hysterical blepharospasm also exists, and in these circumstances the presence of other stigmata of hysteria would make the diagnosis clear.

Blinking in children, which is really a sort of intermittent blepharospasm, may be due to some slight lid irritation, as from a follicular conjunctivitis or to some refractive error, but is very often a habit spasm from which the child will recover in time with attention to his general health. Paralysis of the orbicularis from a seventh nerve lesion is recognized by the position of the lower lid and the patient's inability to close his eye completely. The faulty position of the lower lid leads to eversion of the punctum and epiphora, so that if epiphora is alone complained of, as it often is, it is wise to make sure that the facial nerve is functioning.

The *levator palpebræ* comes under our notice next. Paralysis of the muscle causes ptosis on the side affected and, if due to a complete third nerve lesion, will be accompanied by the other signs of oculomotor paralysis. Ptosis, however, may be present from other causes. The congenital form is recognized as being present from birth, and is usually, though not always, bilateral. It is due probably to a congenital weakness or absence of the levator and can be cured by a suitable operation. A variation of it is the type in which the upper lid is drooped, but rises to a normal position or higher on mastication or lateral jaw movements—the so-called jaw winker. Paralysis of Müller's muscle, the unstriped levator of the upper lid, causes a slight degree of ptosis, and is observed in paralysis of the sympathetic. Spasm of this muscle can be observed on the instillation of cocaine.

Muscles of the Globe.

Their nerve supply, the interference with which leads to various trains of symptoms, makes the ocular muscles of great importance to the neurologist. I could not possibly hope to discuss in this paper the significance and site of the various lesions which may occur, but it might be of value to say something about the nerve supply of the muscles and how one may detect which is involved. Interference with or total loss of its nerve supply will, of course, cause paresis or paralysis of any given ocular muscle.

The third nerve supplies all the ocular muscles with the exception of the external rectus, which is supplied by the sixth, and the superior oblique, which is supplied by the fourth. Any attempt to move the eyes in the direction in which affected muscle acts will result in lack of proper mobility on the affected side, so that the image of the object looked at falls on portions of the macula of each eye which do not correspond, the result being double vision or diplopia, which is the first symptom of an ocular palsy. This diplopia may be slight or absent in certain directions and very pronounced in others. The direction in which it is most marked is the direction of the normal action of the affected muscle. There are cases of diplopia in children in which a latent squint becomes apparent at a comparatively late age. In these the diplopia is present and equal in every direction of the eyes, and there is no lack of mobility observed in any direction. There is always a difference observed by the patient in the two images, one appearing as the real object and the second as its ghost, so that it is very easy to discover which is the affected eye by covering one and asking which image disappears.

In complete paralysis of the third nerve the diagnosis is easy, as in addition to limitation of move-

ment upwards and inwards, there will be a dilated pupil and ptosis of the upper lid.

Paralysis of the external rectus causes loss of outward rotation, which can usually be observed on voluntary effort, and usually these patients have a constant convergent squint owing to over-action of the internal rectus.

In paralysis of the superior oblique muscle there is limitation on looking downwards and towards the paralysed side. In addition to these straightforward conditions, there are many cases in which paresis exists and in which diagnosis is very difficult. It can be arrived at only by a carefully constructed plan of the patient's observation of a suitable object held in the nine principal positions of his field.

Also there are paralyses involving groups of corresponding muscles producing symptoms such as the inability to turn the eyes up or down or to the left or right. The various causes of paralysis cannot be gone into here. The lesion might exist anywhere in the nerve tracts from cortex to muscles. Quite a number of cases I have seen have occurred without any cause being discovered. Syphilis is supposed to be one of the commonest reasons for their occurrence, but I have seen several in which this was not proved, and the condition cleared up with the administration of the time-honoured iodide of potash. *Encephalitis lethargica* has to be thought of in all cases of diplopia, as this symptom may be the first evidence of its onset.

Nystagmus should be looked for during the general examination of the eye. It will rarely be missed if an ophthalmoscope is used, as the finest movements become quite noticeable, especially if the method of starting with a *plus* 20 diopter lens is used. I do not propose to go into its various causes and its significance, except to point out that it may exist in a congenital or infantile form from lack of fixation from visual causes such as congenital defects, high degrees of astigmatism or from albinism, and is a very common condition indeed when one sees numbers of ophthalmic conditions in children.

Concomitant squints form a very large part of the work of an oculist. The parents of children with this condition frequently seek the advice of their doctor in regard to its presence and treatment.

To make a diagnosis as to whether a squint is present and, if so, which eye turns, is very simple. The child is seated or held facing the observer, and an electric light bulb is held above and behind the latter's head. The reflex from this can be seen as a bright spot of light on the child's cornea. If this spot is in the same relative position in both eyes, the child is not squinting. Older children can be told to look at the light. Infants and young children will do so without being told. If the reflex comes from the centre of one cornea and to the outer side of the other, the misplaced reflex comes from the squinting eye, and this eye is turned inwards. A divergent squint would make the reflex come from the inner side.

A latent squint can be seen by getting the child to fix the light, covering one eye with the hand, and then removing it again. If a latent squint is present, the eye will be seen to have assumed a faulty position and to recover itself as soon as the hand is withdrawn.

A condition which is often mistaken for squint in small children is a well-marked epicanthus. Here the wide fold of skin produces an illusion of squint when the child looks to one side, as the cornea of the eye looking inwards meets the inner canthus before it normally should and immediately seems to be squinting. If the test with a light is performed, the eyes are seen to be straight; and as most of these cases cure themselves when the bridge of the nose develops, the parents' fears can be relieved.

When a squint is diagnosed, treatment should be started at once. Never tell parents to wait to see whether the child will grow out of it after teething, or at seven or at some other mystical age, as when this waiting is finished the most valuable time for treatment may have gone. The development of binocular vision continues only up till six years of age, and it is during these early years that most can be done. Also, if the squinting eye is left without treatment for any length of time, its vision fails and an amblyopia, which nothing will remove later, develops. To have only one seeing eye is a tremendout disadvantage, and one which we should do everything in our power to prevent. For this reason I ask you whenever consulted about a child in which you find a squint, to tell the parents to take the child to an oculist or to a general hospital at once.

Lids.

The lids are subject, in common with other parts of the body, to injuries, inflammatory conditions and tumours. With regard to injuries I do not think much discussion is called for here, but there are one or two things that should be mentioned.

First, œdema and extravasations of blood are much commoner and of much greater extent from small causes than in other parts of the body. A slight abrasion or inflammatory change will cause sometimes such swelling of the lids that the eye cannot be opened. Secondly, wounds perpendicular to the lid margin should be brought into correct apposition, as otherwise they will gape and leave a notch, and for this reason incisions made to evacuate abscesses on the lids should be made parallel to the fibres of the orbicularis muscle.

Tumours I shall pass over, except to mention that xanthelasma is a common condition on the lids. It can be removed surgically if required.

Passing on to the lid margins, the common conditions calling for advice and treatment are cases of blepharitis, styes and chalazia.

Blepharitis may be of the scaly or ulcerative type, and may be exceedingly easy to cure or exceedingly difficult. The ulcerative type is distinguished by

the breaking down of hair follicles, followed by abscess formation and subsequently the development of crusts on the lid margin. If these be removed, a small bleeding spot is seen underneath. The lashes are lost sometimes permanently, and the patient's appearance is much disfigured. With regard to treatment, there are two things to be remembered in addition to local applications. First, the patient's general condition must be examined and the general health made as good as possible; and, secondly, it is most important to find out whether any refractive error exists. Children should have a retinoscopy done under atropine and, if necessary, appropriate glasses ordered. The same remarks apply to children or adults who suffer from recurrent outbreaks of styes; all affected persons should be subjected to a careful general examination and the refraction should be estimated. Local treatment in my opinion consists in the removal of the crusts as carefully as possible with a warm solution of bicarbonate of soda, followed by the application of dilute ammoniated mercury ointment mixed with an equal quantity of boracic acid ointment. The method of application of the ointment is very important. It should be carefully massaged into the lid margin with the finger tips twice a day, quite a considerable time being taken to apply it properly. The duration of the treatment should be long, even if the condition apparently clears up, and it is better to go on for about six months, using the ointment once a day to prevent recurrences.

I use exactly the same routine for recurrent styes, and have had more consistently satisfactory results than from the use of vaccines or metallic injections.

Whilst discussing styes, it is well to remember that there are two different varieties. Both are acute infections of sebaceous glands, the first attacking Zeiss or the sebaceous glands of the lashes and producing the external stye, the second is an acute infection of a Meibomian gland and constitutes the internal stye. In the early stages of both, treatment consists of foments followed by evacuation of the pus where it points.

Chalazion, the other common lid condition, does not present at the lid margin, but as it is a chronic infection of a Meibomian gland, it comes under this heading. It produces a small rounded swelling in the tarsal plate which can be felt through the lid and which usually bulges as a purplish-white spot on the inner surface of the lid. It increases gradually in size, and is best dealt with by a crucial incision on its inner surface followed by a complete evacuation of its contents.

The same remark applies to a chalazion as indeed to all chronic lid infections, that the person concerned should have a careful examination of his vision made, and any refractive error corrected with glasses.

Entropion and ectropion of the lids are common conditions, but need not be discussed. Easy to recognize, their treatment is usually admitted to be the province of the ophthalmic surgeon.

Conjunctiva.

The diseases of the conjunctiva are many and varied. The following seem to me to be those which will more concern the general practitioner: (i) Acute muco-purulent conjunctivitis caused by (*a*) the Morax Axenfeld bacillus, (*b*) Koch-Weeks bacillus; (ii) *ophthalmia neonatorum*; (iii) trachoma; (iv) pterygium.

In every case of acute conjunctivitis one should make sure that the disease is limited to the conjunctiva, and that there is no complicating condition such as corneal ulceration or uveal infection. If either of these exists, I do not think the patient should be treated by anyone but an expert. The symptoms of irritation combined with some photophobia and injection of the conjunctiva with abundant muco-purulent secretion are very typical, and if these are combined with a bright clear cornea and an active healthy iris, the condition can be treated with certainty as an uncomplicated acute conjunctivitis.

Of the two forms mentioned here, the angular type is recognized by the redness being mainly confined to the inner and the outer canthus. Koch-Weeks infection, the common cause of conjunctivitis epidemics, produces a more diffuse redness, and is sometimes accompanied by small conjunctival hæmorrhages. A smear will sometimes show what type of infection is present, the bacilli being typical.

The treatment of any type of purulent conjunctivitis is similar, consisting of frequent douching, remembering that the addition of zinc sulphate to the lotion in angular conjunctivitis is a specific and clears up the infection with marvellous rapidity. With regard to the various preparations used as drops, people pin their faith to many. I find mercurochrome more valuable than any, and use it in a 2% solution. I should like to remind people who use silver preparations that argyrosis is a condition to be considered. I have seen it in three patients in the last year, none of whom seemed particularly pleased with the result. I think actually these people had drops repeated over and over again, on their own responsibility, but in any case it is as well to warn them against doing this. *Ophthalmia neonatroum* of gonococcal origin is not a particularly common condition out here; nevertheless preventive measures are important, but I do not think the use of silver nitrate is wise, as it may do damage to the corneal epithelium. If the condition is established and giving a positive smear for gonococci, I recommend its treatment in hospital. The complications are too grave, and the sequelæ of blindness or even total loss of an eye too serious to take any chances with it whatsoever.

Trachoma.

Trachoma is a disease which occupies an important place in the eye work in this country. Its importance to the physician is the recognition of it in its early stages, so that adequate treatment can be started at the most favourable time for a

cure. Its treatment is always tedious, but when it is in an advanced stage the difficulty is enormously increased. Opinions vary as to its most frequent site of commencement; apparently in different countries it does attack different parts first. Out here it is my experience that the conjunctiva overlying the tarsal plates is the commonest site. There may be found only a few regularly placed granules. They are grey in colour and translucent, like grains of sago. A search for them should be made also in the retrotarsal folds. An advanced case is very easy to diagnose, but the early stage may be very difficult, and if any doubt exists it is better to consult an expert. Infection of the cornea sets in very early, and a careful search should be made, especially in the upper portion, with an adequate illumination and magnifying glass for the presence of minute conjunctival vessels which spread into it towards the centre. If there is any doubt about the lid condition, and when vessels are present, the diagnosis is confirmed. Trachomatous pannus when fully established is a gross change easily recognizable, and is very rarely mistaken for anything else.

A short mention of the treatment is all that is indicated here. I pin my faith to expression of the granules followed by the application of copper sulphate over an extended period and drops of a one in one thousand solution of perchloride of mercury. There are, of course, many complications of the disease which require special treatment. The infectivity of trachoma is, I think, established beyond any question of doubt. I saw a child the other day living in the city who developed the disease following the visit of a friend from the North who had been suffering with so-called granulated lids. Practitioners should warn people of possibilities like this, and advise the use of separate towels and soap, wash basins *et cetera*. I am sure that the towel and the wash basin situated outside the back door in so many farms in this country and used by the whole family is often responsible for spreading the disease.

Pterygium.

Pterygium is another very common eye infection in this country which calls for little comment. It is easy to recognize. It may remain stationary for many years, and, if not a marked disfigurement, can be safely left alone. If it frequently gets inflamed or if its head starts to override the limbus and to appear on the cornea, it should be removed.

Lachrymal Apparatus.

The lachrymal apparatus is one which is subject to many disorders. For the purpose of this paper I should like to mention one or two. Infants are commonly affected with dacryocystitis. This may be due to an actual congenital constriction of the naso-lachrymal duct, leading to stagnation of the tears in the lachrymal sac, or to an ascending infection from the nose, which produces the same mechanical effect. In either case it is wise to use some local treatment, such as boracic douching and mercurochrome drops. The drops should be put both into the eye and up the nostril. The contents of the sac should also be evacuated by pressure over it at frequent intervals for a short period, and if this is unsuccessful a small probe should be passed, and this usually completes the cure. To leave these patients for periods up to six months is liable to produce a chronic infection which is extremely difficult to cure. The early signs are definite watering of the eye combined with a little muco-purulent secretion, nearly always limited to one side; this should make one suspect the condition.

Always remember to examine carefully both upper and lower puncta of an irritable watering eye. Foreign bodies and lashes quite often lodge in the puncta and can be seen protruding. Their removal causes a dramatic cure of the symptoms.

Cornea.

Corneal changes do not figure very largely in the general practitioner's work. Two conditions very commonly met with which call for comment are ulcers and abrasions and foreign bodies. Ulcers and abrasions can be easily recognized by instilling a solution of fluorescin, and noting after it has been washed out again with a little boracic lotion, that the area denuded of epithelium is stained bright green. Everyone knows the value of this, but in my experience very few carry it out. With regard to foreign bodies, I would suggest that those who remove them, do so using some adequate form of magnifying spectacles, so as to make sure the foreign bodies have really been removed. If the epithelium is at all damaged in the process, or if the foreign body has been present for some time and the surrounding cornea is infiltrated and hazy, atropine should be instilled and the eye bandaged (provided there is no purulent discharge from the lids) until the spot has healed and the eye becomes white. I consider the usual form of eye spud of value only when the foreign body can be brushed off the surface. When it is embedded to any degree, it can be picked off much more cleanly and effectively with a sharp needle.

The many inflammatory and degenerative changes to which the cornea is susceptible, I do not propose to discuss, except to mention that they are of interest to the physician in that many of them are linked up with a focal infection, the search of which will be in his hands.

The Iris.

Alterations in the pupil and its reflexes, of great importance to the physician, and especially to the neurologist, would involve one in so much description and discussion that I have omitted them. Their detection and application have become in any case more the realm of medicine than of ophthalmology. Changes in the iris itself bring us on to the common ground, in that the inflammatory changes

in this tissue are nearly always (apart from traumatic conditions) the outcome of some systemic infection, and the oculist frequently has to ask for the physician's help in diagnosing and removing that cause. The usual list of teeth, tonsils, sinuses, Neisserian infection *et cetera,* has to be gone over in every case, as, though local medication may result in an apparent cure, recurrences will take place if the cause is not removed.

Though it is strictly speaking not a disease of the iris, I should like to say something about glaucoma and its diagnosis in the acute form. Most of you have been called in to a patient with an acutely painful eye, often in the small hours of the morning, and have had to make a diagnosis of the cause. Acute iritis and acute glaucoma are far and away the most common. The differences between the two are discussed in every text book on ophthalmology, and yet sometimes the diagnosis is hard to make. Leaving the tension out of the question, as it is difficult for unpractised fingers to estimate, and may be raised in both conditions, I think the two most important points to observe are the size of the pupil, dilated vertical oval in glaucoma and small and often irregular in iritis, and the depth of the anterior chamber, very shallow in acute glaucoma and usually normal in iritis. There are many other points of difference, of course, and often the diagnosis is easy, but if it is quite uncertain, and expert help is unobtainable, I advise using a mild mydriatic, such as homatropine and cocaine. In a little while in acute iritis this will dilate the pupil sufficiently to show its irregularity and some posterior synechiæ, and the diagnosis will be made certain. Its effect can easily be overcome by eserine if necessary.

In less acute forms of uveal infection, such as mild iritis, and iridocyclitis, there is always present a certain amount of ciliary injection. Its appearance is typical as a pale violet zone round the cornea in which no individual vessels are seen; and, if any doubt exists as to whether the injection is in the superficial or deep vessels, a drop of adrenaline will be of great help in clearing it up, as it removes the former and leaves a ciliary injection practically unchanged.

Lens changes are very common, especially in the senile form, and can be easily seen by dilating the pupil and examining them with a *plus* lens with the ophthalmoscope. They should never be diagnosed by reflected light as the pupils of most elderly people look grey, even though the lens may be quite transparent to transmitted light. Their course is usually extremely slow and in some cases they do not appear to change from year to year. Their medical treatment is of little use. Thyreoid has been tried and the removal of septic foci, while of course in cases due to diabetes proper treatment for this condition may be a considerable help. Opacities in the vitreous are often signs of auto-intoxication and, if present, even without any other symptoms, are an indication for a general examination.

Fundus Changes.

With regard to fundus changes, a huge field is opened up. I can touch only the fringe of it by describing what I think are the important things to look for and the changes in a few well-known diseases. Everyone using an ophthalmoscope looks first for the optic papilla and uses it as a landmark, and also very often as a means of measurement of other conditions in the fundus. The disk itself is about 1·5 millimetres in diameter, usually circular or slightly vertically oval, and its appearance varies in health enormously in contour, colour and clearness of outline. Also the distribution of the vessels in and upon it are subject to great variety, and it is only by observing numbers of normal disks that an appreciation of what constitutes a pathological condition can be arrived at. Before going on to a description of some pathological conditions, it might be well to consider what one is observing in a normal fundus. The so-called optic disk is really a hole in the continuity of the sclerotic and chorioid bridged across by the *lamina cribrosa,* through which the fibres of the optic nerve pass to be distributed over the retina. What one sees then at the edge of the so-called disk is an inner or white ring which is composed of the sclerotic fibres which lie between the margin of the chorioid and the optic nerve, and, secondly, a pigmented ring which varies tremendously in its density and marks the edge of the chorioidal aperture. These anatomical facts have to be borne in mind in considering the conditions such as papillœdema in its early stages.

The blood vessels of the optic nerve, which are central before reaching the globe, divide at the head of the nerve into a number of branches, both arteries and veins. The arrangement and point of division of these vessels vary between wide limits, but there is always a division into upper and lower temporal and nasal branches which can be made out.

In a healthy eye the arteries are bright red with a central white streak or reflex running along them. the veins are larger, slightly more tortuous and are distinctly darker in colour.

The division of the nerve fibres forms a hollow cone or cup, the so-called physiological cup at the bottom of which the *lamina cribrosa* can often be seen as a white area covered with small greyish-white spots. The blood vessels appear on the inner border of the physiological cup. Owing to the course which the nerve fibres take, the outer or macular portion at the disk is thinner than the inner and for this reason the outer side of the disk is always paler even in normal eyes.

With regard to pathological conditions in the fundus, those of most importance to the physician I should say are: (i) Arteriosclerotic changes, (ii) albuminuric neuroretinitis, (iii) diabetic retinitis. (iv) papillœdema, (v) embolism and thrombosis of retinal vessels, (vi) optic atrophy.

My own experience of arteriosclerosis is that the condition has usually been diagnosed clinically

before the eye changes are taken into account. Rarely I see patients exhibiting early changes who are not under medical attention and who can be referred to their own physician for advice. The earliest signs to look for are loss of translucency in the retinal arteries, faint white lines at the margin where vessels intersect and displacement of the retinal veins in the direction of the blood stream in the same direction. The last mentioned sign appears before actual nipping of the vein, which occurs only when the condition is advanced; later changes, such as hæmorrhages, narrowing or complete occlusion of the vessels with "copper wire or silver wire arteries", occur when the disease is fully established.

I have been amazed on several occasions when I have been able to reexamine fundi in which every possible arteriosclerotic change is present and multiple hæmorrhages, to find how the hæmorrhages will disappear and the fundus picture improve after the patient has been put on a suitable medical *régime*.

Albuminuric neuroretinitis is usually a very well defined and easily recognizable condition. The presence of the star-shaped white patches in the macula combined with a swollen disk and œdematous retina makes it unmistakable. It is commonest in chronic interstitial nephritis, and is always a grave prognostic sign. Associated with pregnancy it is an indication for induction of labour in the early stages, but the later it appears, the less serious is the prognosis. There is always some loss of vision following it; sometimes a great deal of permanent damage is done.

Diabetic retinitis is not nearly so common. The main points of difference from the albuminuric form are that the hæmorrhages, if present, are round rather than flame shaped and often the vessels and optic disk are normal. If albuminuria exists in conjunction with diabetes, the fundus change may be typical of that form.

Papillitis and papillœdema are very important diagnostic signs. Their presence in a marked degree is very easily seen, and the difference in level between the swollen disk and the remaining retina can be measured.

There are many pitfalls, however, in its detection in its early stages. The two names mean two distinct pathological changes. The first, papillitis, is an inflammation of the nerve head, often the sequel to a descending infection of the optic nerve, such as may be caused by a meningitis; the second, papillœdema, is an œdema from back pressure, such as may be caused by a cerebral tumour. Unfortunately, the two types merge clinically into one, and it is often impossible to differentiate them.

Broadly speaking, the inflammatory type is accompanied by redness of the disk and often inflammation of the surrounding retina, while in papillœdema the swelling is more sharply defined from its surroundings and the nerve head is paler. However, in intracranial diseases it is possible to get a papillœdema combined with exudates in the macula which simulates the star-shaped figure of neuroretinitis, so that the distinction is not always possible and the diagnosis has to be made on accompanying physical signs.

The first sign of papillœdema is blurring of the disk margin, usually on the nasal side, followed by blurring of the whole circumference. The arteries become smaller, the veins are distended. Later a measurable swelling of the disk may be seen, and exudates make their appearance and often hæmorrhages into the disc or immediately surrounding it.

The conditions which simulate it must be recognized. Astigmatism is one. In a highly astigmatic person it is impossible to focus both diameters of the disk at the same time, but by careful focusing up and down, the previously blurred portion will become sharp, while the rest gets blurred. If the patient has glasses which correct his astigmatism, it is a good idea to view the disk through them; and this can be done by tilting them slightly to one side to get rid of the central reflex, when the whole disk will become sharp and distinct. Highly hypermetropic people often appear to have blurred disks, the so-called pseudo-papillitis. In these, although the disk appears at first sight exactly like a true papilllitis, no swelling can be detected. It is my own rule in a doubtful case not to diagnose the condition unless a definite amount of swelling can be seen, and also to make frequent examinations to watch for changes which may occur quite rapidly and make the diagnosis certain. Another point in connexion with papillœdema is that quite good vision, even $^{6}/_{6}$, may exist with considerable degrees of swelling.

Embolism and thrombosis of retinal vessels are of interest to the physician from their connexion with other vascular changes. When occurring in the central artery or vein, they are, I should say, the two commonest causes of sudden loss of vision in one eye. When they affect the central artery, the picture of embolism is just what we would anticipate—a total emptying of the retinal arteries, which appear as threads, while the smaller vessels disappear altogether. The macula becomes blanched, the fovea alone maintaining its red colour and appearing by contrast as a bright red spot in the centre of the white area. The future vision in these cases is practically nil.

Thrombosis of a retinal vein occurs in people who have high blood pressure, often albuminuria and vascular degeneration. If the central vein is affected, the whole retina becomes a mass of hæmorrhages of every shape and size. The veins become distended and distorted, while numerous exudates make their appearance. Loss of vision is invariable in these cases, while the eye is very liable to undergo changes leading to secondary glaucoma.

Optic atrophy is of two types, primary and secondary. The former is recognized by a progressive pallor of the disk, going to paper whiteness,

the edges of the disk keeping their distinct outline. In the secondary type which follows such conditions as albuminuric neuroretinitis, papillœdema *et cetera*, the disk appears pale but quite flat, and its edges, owing to the presence of fibrous tissue formation, is blurred in outline. In its primary type the vessels may appear normal or only slightly narrowed, while in the secondary type there is usually definite narrowing.

Primary optic atrophy is commonest in tabes, and, if established, usually goes on to complete blindness. It may be present in disseminated sclerosis also, and in this disease the prognosis as regards vision is distinctly better. In making a diagnosis of optic atrophy, the normal pallor of the temporal part of the disk should be taken into account, and not confused with the pallor of its true condition, which is general and progressive. A large physiological cup may also be mistaken for a pale disk if care is not taken to notice that it is surrounded by a line of normal disk colour.

Primary atrophy is usually accompanied by slight cupping of the disk, which is saucer-shaped, and quite different from the cupping of glaucoma, which is sharply defined and even overhanging. The vessels in the latter are seen apparently to stop and then to reappear at a higher level at some distance away from their original course.

In conclusion, I should like to repeat that the normal disk varies widely in its colour and unless meant to refer to a pathological condition, I think such descriptions as "slightly pale", "showing slight pallor" should not be used. Either the optic papilla is undergoing a pathological change or it is healthy, and a report such as this, perhaps ending with fundus "otherwise normal", is quite meaningless.

Herpes Zoster.

Herpes zoster ophthalmicus should be mentioned, as it is not an uncommon condition, and one in which the general practitioner is usually called upon for advice. The skin lesions call for no special comment, but I should like to stress the importance of the eye complications if they occur. I have seen practically every variety and degree of corneal involvement from a superficial vesicle to a deep septic ulcer. The iritis and iridocyclitis may occur independently of the corneal complications, and I do not think that at any time during the course of the disease one can say that the patient is safe from them. As the lids are swollen in most cases and the eye difficult to see, and the pain of the herpes is difficult to differentiate from the pain of an eye complication, I think these patients should all be seen by an oculist in case special treatment is necessary. After the acute symptoms have subsided and the patient is able to open his eye again, it is disconcerting to find that his vision is much reduced from a keratitis which has passed unnoticed. The cornea becomes insensitive as a result of the herpes, and even a deep-seated involvement of it causes no pain.

SALPINGOSTOMY.[1]

By Cecil Coghlan, M.B., Ch.M. (Sydney), F.R.C.S. (England), F.R.C.P.I., D.G.O. (Dublin), F.R.A.C.S.,

Honorary Assistant Gynæcologist, Saint Vincent's Hospital; Honorary Gynæcologist, New South Wales Community Hospital, Sydney.

During the investigation of a series of cases of sterility of the human female, both Fallopian tubes are found in some instances to be impermeable to the flow of gas in intrauterine insufflation, even at a pressure as high as 200 millimetres of mercury. Further investigation by means of X rays (utero-salpingography) discloses the position and nature of the blockage. Can we do anything to help these women to gain their desire—conception and the birth of a live child? If not, they are condemned to a lifelong sterility, knowledge of which will probably cause them much mental anguish. If the husband is aware, too, that his wife cannot bear children, it may lead to much domestic disharmony. On these grounds it behoves us to do anything we can, provided it holds out some hope of success.

Selection of Cases for Operation.

This brings us to the consideration of the type of patient on whom we may perform the operation of salpingostomy. Figure I illustrates an ideal case; the body of the tube on either side is regular and smooth, the ampulla is somewhat dilated, and on the right side the umbilication where the fimbriæ have been invaginated and become adherent, can be seen. On the left side the ampullary end is not completely closed, but is shut off from the general peritoneal cavity by adhesions. This is ascertained by the distorted appearance of the end of the tube, indicative of adhesions; by the failure of the gas to pass on insufflation; by the freedom of the abdominal cavity from oil as discovered by X ray examination twelve hours later. On both sides, when the radiographs are viewed in the stereoscope, there is evidence of peritubal adhesions. The history in this case discloses a mild puerperal infection following the birth of a child eleven years previously, since when no further conception has occurred. In Figure II is shown the appearance after operation, with both tubes freely open and oil droplets in the abdominal cavity.

The causes of tubal occlusion are: puerperal sepsis, full-time or post-abortional; gonorrhœa; pelvic peritonitis following appendicitis *et cetera*; pneumococcal peritonitis (often unrecognized during a pneumonia); mild pelvic peritonitis associated with the acute infectious diseases (measles, mumps, scarlet fever *et cetera*); tuberculosis.

Puerperal sepsis results in a picture more or less similar to that described above, and is fairly favourable for salpingostomy.

Gonorrhœal infection frequently occludes the tubes at the uterine ends or causes tubal irregu-

[1] A preliminary communication with the report of two intrauterine pregnancies following operation.

ILLUSTRATIONS TO THE ARTICLE BY DR. CECIL COGHLAN.

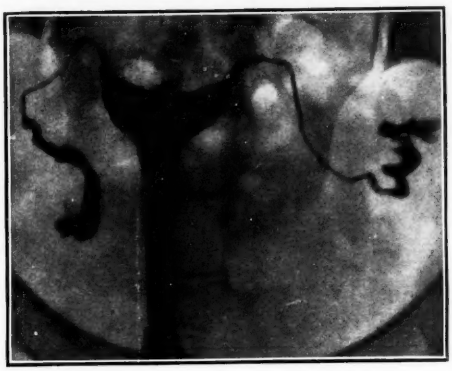

FIGURE I.

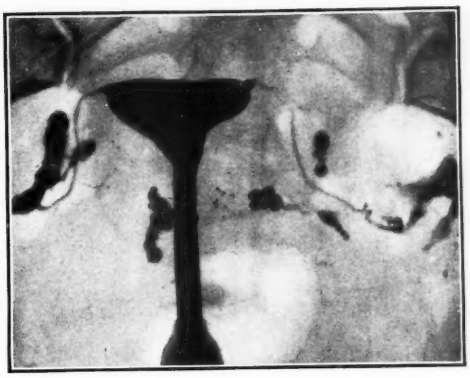

FIGURE II.

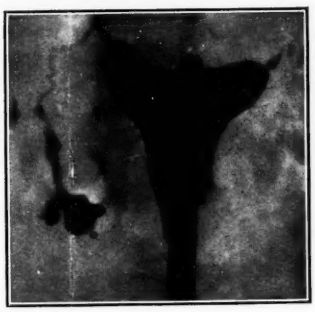

FIGURE III.

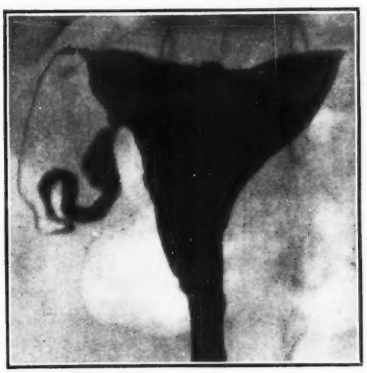

FIGURE IV.

FIGURE V.

FIGURE VI.

FIGURE VII.

ILLUSTRATIONS TO THE ARTICLE BY WILFRID BISHOP.

FIGURE I.

Cholesterol radiated in chloroform solution with 10 milligrammes of radium in platinum needles. Magnification 100. Note almost entire absence of crystalline form. The drop formation is characteristic of the resinous substance left after prolonged radiation.

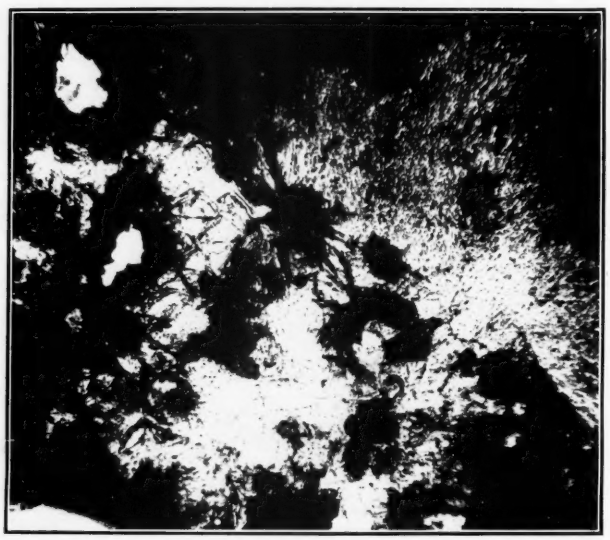

FIGURE II.

Cholesterol from control chloroform solution. Not radiated. The same volume, and hence the same amount of cholesterol, was used as for Figure I. Magnification 100. Note dense crystal structure and absence of drop formation.

larities and sclerosis of the muscular tissue of the tubal wall. If this is annular, the contraction of the fibrous tissue occludes the tube in its length. These conditions are shown in Figure III, where, on the left, there is an annular constriction closing the tube in its length, and on the right, the tube is badly kinked by adhesions, the lumen is irregular and the ampulla distorted and closed. This type of infection is not at all favourable for plastic work on the tubes.

Pelvic peritonitis rarely infects the interior of the tubes; it causes closure of the fimbriated ends and kinking due to peritubal adhesions. Operation here is very favourable.

The same remarks apply to pneumococcal peritonitis and that associated with the exanthemata as to pelvic peritonitis described above, but with this difference: the tubes are rarely kinked and the adhesions are usually light.

In tuberculosis the tube becomes sclerosed and it is impossible to do anything with it.

In those cases in which a salpingograph discloses that the tubes are closed but not severely damaged, salpingostomy can be performed with some hope of success. The ideal is the club-shaped one.

The technique I use in the operation I have never seen published, so I will give it later in detail. I have used it now in eleven cases; in ten of these a salpingograph taken about a month after the operation reveals that the tubes treated are now open. One patient, immediately on leaving hospital, contracted a diplococcal infection, and I was unable to take a salpingograph for nine months. This disclosed that both tubes were then blocked at the isthmus, although previously they had been open to the ampullary ends. This can hardly be classed as an operative failure.

Results.

Of the ten patients on whom operation was successfully performed, one, operated on in April, 1929, conceived *in utero* and bore a healthy child in July, 1930. The mother had been married eleven years and had had a septic abortion the year following her marriage.

A second patient, operated on in March, 1930, is now (October, 1930) ten weeks pregnant. In this case there had been a previous pregnancy, terminated by craniotomy and followed by puerperal sepsis. The right tube was closed and greatly kinked by adhesions, and the left tube was sclerosed. The right tube only was opened at the operation; the left was removed. Figure IV shows the condition before operation and Figure V after operation.

The Operation.

The detailed technique of the operation I use is as follows. The abdomen is opened, a thorough inspection is made of the pelvic organs, and all adhesions around the uterus, tubes and ovaries are carefully separated. The Fallopian tubes are then taken in turn and fixed with a stay suture on each side towards the ovarian end. An incision is then made with a knife through the serous and muscular coats only. The incision extends about one-sixth of the length of the tube and is situated opposite the attachment of the mesosalpynx; the lateral end of the incision extends to the free edge of the mesosalpynx. The mucous membrane is now opened in the middle of the exposed area with a fine pair of scissors, and the opening extended either way until just short of the ends of the sero-muscular incision. Any bleeding can usually be stopped by the application of a hot pad, which is left in contact with the cut area while the other tube is being opened in a like manner. Interrupted sutures of fine catgut on a fine, curved, non-cutting needle are inserted in and out through the mucous membrane, and again in and out through the seromuscular coat, the sutures being inserted perpendicular to the cut edge. The sutures placed this way, when tied, roll out the mucous membrane over the cut edge of the sero-muscular coat and roll in the cut edge of the mucous membrane, thus leaving no exposed raw areas. I use just as few sutures as are necessary completely to evert the mucous membrane. The details of the operation are shown in the diagrams.

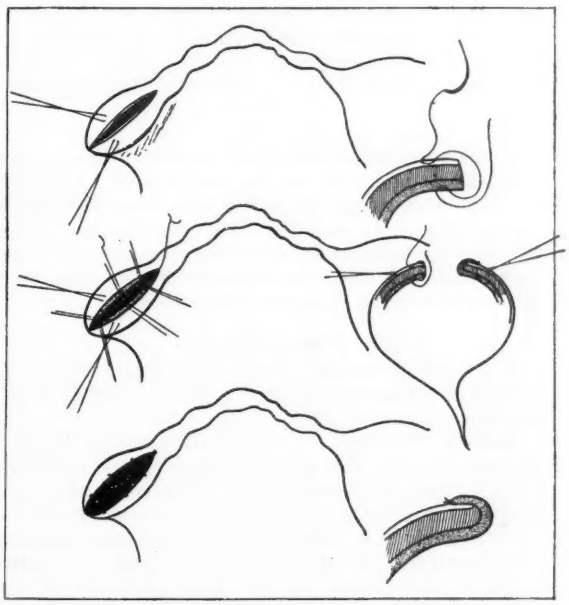

Diagram showing steps of the operation.

Figure VI beautifully shows the opened end of a tube after this operation, with the lipiodol adhering to the mucous membrane of the new ostium and free in the abdominal cavity. The other tube had been removed by another surgeon at a previous operation. Figure VII shows the condition in the same patient before operation.

Eleven is a small series, but as I do the operation only in selected cases, it will take some time to build up a series of reasonable dimensions. The results to date, I think, justify publication; later, I hope to give details of a much larger number.

Postscript.

This paper was written in October, 1930. From then until the end of 1931 I performed a further six salpingostomies, all of which have remained patent, bringing the series up to seventeen cases. A third patient became pregnant and I delivered her of a live child in January of this year. On the second patient who conceived, reported in the paper, I performed a Cæsarean section in April, 1931, delivering a live child, and had an opportunity of examining the opened tube. It lay free, with no adhesions around it.

Summary.

Salpingostomy is a successful procedure in picked cases of tubal occlusion. In the paper and postscript seventeen cases are reported, with three pregnancies, each of which was brought to a successful conclusion. Salpingographs taken before and after operation are shown. The operation is described in detail.

THE ACTION OF X RAYS AND THE RAYS FROM A RADIUM NEEDLE UPON CHOLESTEROL IN VITRO.[1]

By WILFRID B. S. BISHOP, M.Sc., A.A.C.I.,
Bosch Cancer Research Fellow.
(From the Department of Physiology, The University of Sydney.)

THE experiments described in this note are preliminary to a study of the reaction of blood, red cells in particular, to radiations from radium in various metal containers. The experiments which follow were made since there are so many published results at variance, some authors stating that there is no action when cholesterol is radiated.

Previous Work.

It is not proposed to give a detailed survey of earlier experiments concerned with cholesterol and short wave radiations.

Roffo and Correa,[8] using rat tumours, found a decrease in the cholesterol content after radiation with X rays, the concentration being reduced by one-half in three hours. These authors also report *in vitro* experiments; they used a chloroform solution of cholesterol. With 200 kilovolts and four milliampères at a distance of seventeen centimetres, and no filter, there was a complete disappearance of the cholesterol after eighteen minutes' radiation. The authors also state that the X radiation had no effect on chloroform alone. These results were in part confirmed by other workers. Thus Dognon,[3] Reinhard,[7] Bachem and MacTate,[1] Burgheim[2] and Hieger[4] state that the cholesterol is destroyed, but only in halogenated solvents, as chloroform or carbon tetrachloride, and is not affected in benzene, alcohol and the like. The general conclusion appears to be that the action of short wave radiations on cholesterol is indirect and, when present, is due to a prior action on the solvent.

Roffo[9] claims that the reaction proceeds in benzene, while Reinhard and Buchwald,[6] using chloroform and ethyl alcohol, report positive results with both solvents. Schreuss and Schulze[10] state that cholesterol is not affected in any solvent by radiation. This work has been repeated by Roffo, but the results were not confirmed.

Experiments with X Rays.

A solution of cholesterol was placed in a thin-walled glass tube (0·5 centimetre in diameter) and the tube was sealed in a blow pipe flame. The tube was then exposed to X rays for sixty minutes, under the following conditions: Tube current, 140 kilovolts; tube potential, 10 milliampères; distance of solution from target, 34 centimetres. No filter was used.

I am indebted to Dr. W. Moppett for his kindness in making these exposures and for the details of the conditions employed.

The small tubes, being completely sealed, protected the cholesterol solutions from any nitrogen oxides which may have been present in the X ray room. The results are given in Table I.

TABLE I.

Action of X Rays on Cholesterol Solutions. Radiation conditions as given above.

Solvent.	Cholesterol in grammes per 100 Cubic Centimetres.	Depth of Solution.	Melting Point.		Decrease in Strength of Solution.
			Irradiated.	Control.	
Chloroform ..	0·50	35 millimetres.	106° to 111° C.	145° to 146° C.	54%
Benzene ..	0·50	35 millimetres.	145° to 146° C.	145° to 146° C.	Nil.
Ethyl Alcohol..	0·50	35 millimetres.	145° to 146° C.	145° to 146° C.	Nil.

The residue after evaporation of the chloroform was found to be a brown resinous mass which had a much lower melting point than the original cholesterol (126° to 131° C., as compared with 146° C.). With benzene and ethyl alcohol no reaction was observed.

Technique.

All the solvents in both X ray and radium experiments were dried and fractionated. The cholesterol used was prepared by British Drug Houses, Limited (London), and had a melting point of 142° to 146° C. After three recrystallizations from absolute alcohol the melting point was 145°–146° C. This material was used in all the experiments reported here.

As the main interest lay in the action of radium radiations on cholesterol, further experiments with X rays were not carried out, particularly as these three experiments show that the reaction does take place and that it is apparently dependent on the solvent. The cholesterol solutions were made up and the radiation made in a fifty cubic centimetre

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney, and with the aid of the Cancer Research and Treatment Fund. Submitted for publication September 3, 1931.

glass stoppered flask, fitted with a mercury seal. This was found necessary to prevent loss of solvent by evaporation when the exposures were lengthy. The seal was conveniently made by slipping a short length of rubber tube over the neck of the flask so that it projected above the top of the stopper. The stopper was inserted and the space filled with mercury.

The radium, in platinum and Monel metal containers, was placed in a thin-walled tube, constricted some distance from the bottom, so that the radium was centrally placed when the tube was immersed in the cholesterol solution.

When required, one cubic centimetre samples were withdrawn from the radiation and control flasks and the cholesterol estimated by Liebermann's method. Before this method could be applied to the alcoholic solutions, the alcohol had to be evaporated.

Experiments with Radium.

The radiations were carried out in the flasks as previously described, except for one series, which was also carried out in a tube approximately 2·5 centimetres in diameter.

The action of the radium radiation on the solvents was first tried. With chloroform and carbon tetrachloride the presence of free chlorine after radiation was shown on the addition of potassium iodide and starch solution by the liberation of iodine with the formation of a blue colour. This was not given by the iodide-starch solution alone.

The boiling points of the chloroform and the carbon tetrachloride were lowered nearly 1° C. after radiation, while those of the benzene and ethyl alcohol were not altered. This is not in agreement with the findings of Roffo, but does agree with Heiger and Dognon.

The experiments with cholesterol were made with solutions of two concentrations, one and five milligrammes per cubic centimetre. With the five milligramme solutions no reaction could be detected in any of the radiated samples, irrespective of the solvent used. The experiment was repeated in tubes so that the thickness of the solution was nine to ten millimetres around the radium tube. The reaction was then found to take place as for the dilute solutions (Tables II and III).

In each group of experiments the cholesterol in chloroform and carbon tetrachloride underwent a change (only more slowly), similar to that produced by X rays.

The change to a resinous substance, which cannot be recrystallized, is strikingly shown in the photographs, the tendency of the irradiated cholesterol to form droplets being quite characteristic and distinct from the familiar crystalline structure. (See Figures I and II in supplement.)

Summary.

1. Radium radiations from platinum and Monel metal containers liberate chlorine from chloroform and carbon tetrachloride and lower the boiling points approximately 1° C.

Table II.

The Action of Radium Radiations upon Solutions of Cholesterol.

Grammes of cholesterol per 100 cubic centimetres of solution: 0·100 gramme.
Depth of solution: 4·00 centimetres.
Radium used: Two 5·0 milligramme platinum needles.

Solvent.	Time of Exposure.		Strength of Cholesterol Solutions.		Percentage Decrease in Strength.
	Days.	Millicurie Hours.	After Radiation.	Before Radiation.	
Chloroform	3	720	0·098	0·100	2·00
	7	1,680	0·095	0·100	5·00
	10	2,400	0·092	0·100	8·00
	22	5,280	0·080	0·100	20·00
	28	6,720	0·063	0·100	37·00
	38	9,120	0·047	0·100	53·00
Carbon Tetra-Chloride.	3	720	0·096	0·100	4·00
	7	1,680	0·093	0·100	7·00
	10	2,400	0·088	0·100	12·00
	22	5,280	0·078	0·100	22·00
	28	6,720	0·061	0·100	39·00
	38	9,120	0·035	0·100	65·00

Benzene, Ethyl Alcohol: No reaction detected at all.

Table III.

The Action of Radium Radiations upon Solutions of Cholesterol.

Grammes of cholesterol per 100 cubic centimetres of solution: 0·500 gramme.
Depth of solution: 4·00 centimetres.
Radium used: 20 milligrammes in Monel metal tubes.

Solvent.	Time of Exposure.		Strength of Cholesterol Solutions.		Percentage Decrease in Strength.
	Days.	Millicurie Hours.	After Radiation.	Before Radiation.	
Chloroform Carbon Tetra-chloride Benzene Ethyl Alcohol	No reaction detected over fifty-four days.				

Depth of solution: 10 millimetres in sealed tube.

Chloroform.	1	480	0·490	0·500	2·00
	2	960	0·480	0·500	4·00
	4	1,920	0·450	0·500	10·00
	9	4,320	0·410	0·500	18·00
	13	6,240	0·406	0·500	18·80
	18	8,650	0·371	0·500	25·90
	25	12,000	0·300	0·500	40·00
	29	18,710	0·210	0·500	58·00
	54	25,950	0·110	0·500	78·00
Carbon Tetra-chloride.	1	480	0·490	0·500	2·00
	2	960	0·480	0·500	4·00
	4	1,920	0·450	0·500	10·00
	9	4,320	0·400	0·500	25·00
	13	6,240	0·350	0·500	30·00
	18	8,650	0·325	0·500	35·00
	25	12,000	0·250	0·500	50·00
	29	18,710	0·200	0·500	60·00
	54	25,950	0·075	0·500	85·00

Benzene, Ethyl Alcohol: No reaction detected over fifty-four days.

2. Radium radiations from platinum and Monel metal containers reduce the cholesterol concentration when chloroform or carbon tetrachloride is used as a solvent, but do not cause any alteration of the strength when benzene or ethyl alcohol is used.

Acknowledgements.

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VASCULAR DISEASE, WITH SPECIAL REFERENCE TO DIABETES MELLITUS.

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THROUGHOUT the history of medicine there occurs from time to time an alteration in our conception of the processes of a disease and of the relative significance of these processes one to the other. Facts which at one time appeared of little or no significance assume importance in the light of further knowledge, whilst others previously dominant fade more or less into obscurity.

The association of *diabetes mellitus* with vascular disease has been recognized for many years, but as yet the exact relationship is not fully established. In time past, diabetic arteriosclerosis was regarded as a probable exaggeration by this disease of the tendency of mankind to vascular degeneration with advancing years. Within the past decade, however, the introduction of insulin into the treatment of *diabetes mellitus* has produced a marked change not only in the results of treatment, but in the appearance of sequelæ. The occurrence of vascular disease in adolescent diabetic patients, whose lives have been prolonged by insulin, is of much importance, as previously such patients invariably died rapidly from coma before vascular degeneration could become manifest.

It would seem that with the prolongation of the lives of diabetic patients by the modern methods of treatment there is a greatly increased liability of these patients to the development of vascular disease. Joslin(1) has recently shown that arteriosclerosis, as a cause of death in *diabetes mellitus*, has risen from 15% in the pre-insulin era to 47% since the introduction of insulin, and this figure is still increasing.

Thus the problem of diabetic arteriosclerosis, which a few years ago was of comparatively little importance, is now assuming a prominent position in the course of this disease; and it would appear that it will become of even greater significance in the future until some further advances of knowledge throw more light on the ætiology of *diabetes mellitus* and upon the initial lesions of arterial degeneration.

Clinical Considerations.

In any clinical consideration of vascular disease in living subjects it is at once apparent that no methods of examination at present exist whereby the earliest manifestations of vascular change can be recognized. Such methods as are available are of value only in detecting the more advanced degrees of arterial damage. In consequence, any attempt to determine the frequency of this complication in diabetic patients must of necessity under-estimate it.

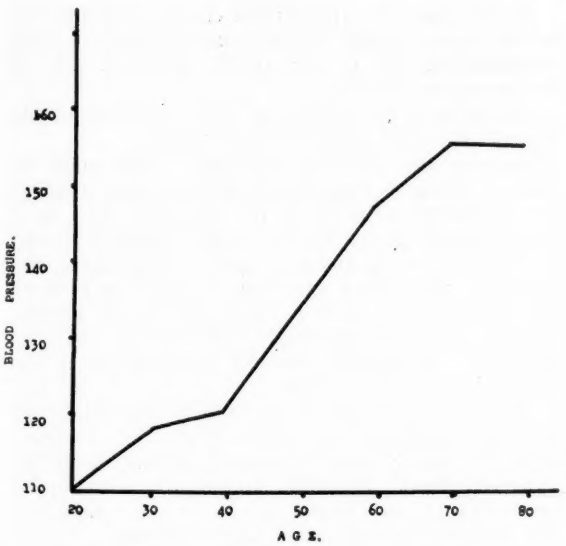
Of the available methods of investigation, the following are worthy of consideration:

1. *Observations of Vascular Thickening and Tortuosity.* The difficulty of establishing any standard of normality or abnormality renders vascular palpation of little value. Warren(2) has rightly said in criticising this method that "it is as old as it is uncertain".

2. *Radiological Changes Observed in the Arterial Wall.* When calcification occurs in an arterial wall the change can be recognized in an X ray plate. This method has found favour in America, and large series of figures based on radiological evidence have been published by Morrison and Bogan,(3) Rühl(4) and others. It is well known, however, that gross arterial disease may exist for a long period without the occurrence of any calcification and, in consequence, this method is of limited use. Statistics based upon radiological appearances alone must of necessity under-estimate the frequency of vascular disease.

3. *Variations Observed in Blood Pressure.* Observation of the systolic blood pressure in diabetic patients reveals the interesting fact that hypertension is not a pronounced feature. This fact has been noted by Joslin(5) in a recent communication. The accompanying curve represents the average systolic blood pressures of three hundred diabetic patients admitted to the Alfred Hospital between 1925 and 1930, graphically expressed against the age in decades. In another group of fifty cases, observation of the systolic and diastolic blood pressures revealed no marked hypertension. An attempt was made to correlate variation in the blood pressure readings with changes in the retina, but no definite connexion could be established between observed vascular damage in the *fundus oculi* and blood pressure.

4. *Changes in the Fundus Oculi.* Ophthalmoscopic examination is probably the most exact single method known at present for the detection of vascular disease. Accepting the views of Foster Moore(6)(7) with regard to the vascular origin of retinal exudates, much valuable knowledge can be



obtained by the study of the diabetic fundus. With the help of Dr. J. Ringland Anderson, fifty diabetic patients were investigated recently by the writer. These patients were selected at random from the diabetic clinic of the Alfred Hospital, their ages ranging from twenty-six to seventy-six years. Of this group of patients, the ocular fundi were found to be normal in twenty-eight and some abnormality was detected in twenty-two. As a result of observation of all these patients over a period of many months it was possible to divide them into two groups: (*a*) Those in whom the diabetic condition had been satisfactorily controlled (by diet and where necessary with insulin also) since the onset of the disease. (*b*) Those in whom the diabetic condition had not been so controlled during the course of the disease.

The criteria adopted of adequate control in these two groups comprised frequent observations of the blood sugar, frequent urinary examinations and a general consideration of the honesty, intelligence, and circumstances of each patient. According to these standards, thirty were considered to have been adequately controlled since the onset of the disease and twenty were classified as uncontrolled. Considering, then, the occurrence of retinal changes in the two groups, the results of examination may be expressed in the accompanying table.

The importance of adequate control of the diabetic state in relationship to the development of retinal changes is strikingly apparent.

5. *Consideration of the Late Results of Vascular Disease in Diabetics.* The onset of vascular disease is unheralded, the course is slow and insidious and so it happens that frequently the disease declares itself after many years with dramatic suddenness in the form of gangrene of an extremity or of coronary infarction.

Of three hundred diabetic patients admitted to the Alfred Hospital between 1925 and 1930, 37 suffered from gangrene of an extremity, 17 suffered from myocarditis associated with coronary disease, 12 suffered from associated chronic nephritis and eight suffered from other vascular accidents, such as cerebral thrombosis. The sexes were almost equally affected in this group.

It is at once apparent that the principal incidence of the vascular lesions occurred in the peripheral arteries and in the coronary arteries. This is an unusual distribution and is in definite contrast to the incidence of vascular lesions which occur in other forms of arteriosclerosis. This is probably explained by the nature of the vascular change which occurs in these cases and by the absence of hypertension. The figures quoted above are in agreement with a series published by Joslin[8]; and Warren,[9] in analysing Joslin's results, states that "the relative immunity of the brain of diabetics to arteriosclerotic injury is due to the relatively small amount of hypertension in this group".

The time factor in the development of vascular changes is a difficult one to assess. It is probably a matter of several years before definite vascular damage occurs. Joslin[10] has stated:

> Exposure of a patient to the influence of diabetes was not long enough in Naunyn's time to show whether it would result in arteriosclerosis. The average duration of the disease was then only 4·7 years and the few diabetics who exceeded this limit were the mild old cases who already presented, at the onset of diabetes, the vascular changes which were due to their age.

The age of the patient is also of importance. During the past eighteen months the writer has had the opportunity of examining at autopsy three young adults, all of whom were under the age of twenty. Each of them had suffered from severe *diabetes mellitus* for over four years, and none of them had been adequately controlled during life. The examination of the arteries of these patients

Table Showing Number of Patients with Retinal Changes.

Group of Patients.	Average Age.	Fundi.			Range of Duration of Disease in Affected Cases.	Average Duration of Disease in Affected Cases.
		Normal.	Arterio-sclerosis.	Retinitis.		
30 Controlled Patients	53·1	24	2	4	3–5 years.	4·0 years.
20 Uncontrolled Patients	59·4	3	2	15	2–8 years.	4·6 years.

disclosed definite evidence of vascular disease in each instance.

It would appear, therefore, that the incidence of vascular disease in the diabetic patient is intimately concerned with the duration of the disease and the adequacy of control by treatment, and that the age of the patient is of secondary importance.

Pathological Considerations.

The pathology of the vascular change in *diabetes mellitus* has received but scant recognition in the literature. Buerger[11] states that the pathology of arteriosclerosis and of diabetic vascular disease is the same and in general such authors as do mention the relationship of diabetes and arteriosclerosis appear to share the same opinion. It is only recently that the predominant involvement of the muscular arteries of the diabetic in arteriosclerotic change has been stressed by Warren.[12]

As a basis for the present paper material from twenty-two patients suffering from *diabetes mellitus* has been examined, and from the changes observed there appears to be ample evidence to support the view of Letulle[13] that certain appearances hitherto thought to be characteristic of other specific forms of arterial disease, such as *thrombo-angiitis obliterans*, are at times to be found in the arteries of diabetics.

Consideration of the Findings in the Three Adolescent Diabetics.

Consideration may be given to the findings in three adolescent diabetics already mentioned. Generalized evidence of vascular damage was present in all three, affecting the larger arteries, both elastic and muscular. The changes appeared to be of the same type with due allowance for the difference in the structure of the two types of vessel.

The lining membrane of the *tunica intima* showed no change *per se*. The intimal layer itself appeared to undergo a patchy fatty infiltration and degeneration which was often localized to one portion of the vessel wall, but which was at times apparent around the entire circumference. The cells of the intima in such areas seemed swollen and stained poorly. There was an apparent increase in the intercellular substance. A later stage showed disintegration of the cells which become replaced by fat. In some areas the primary change appeared to commence in the deeper layers of the intima, while in others the change seemed to be uniform throughout the layer. The nature of the fatty change was investigated by suitable staining methods. In general, the change was a mixed one, lipoids and saturated fats occurring diffusely throughout the degenerating areas.

In the muscular arteries evidence of fragmentation of the *lamina elastica interna* was found in all three cases. Occasionally the lamina appeared to be reduplicated. This change usually appeared in association with fatty change in the intima.

In two cases no appreciable change was detected in the *tunica media*. In one some evidence of fatty degeneration was present in the media of some of the muscular arteries.

No appreciable alteration was observed in the *tunica adventitia* of any of these cases.

Distribution of These Changes. The main incidence of the changes described above was apparent in the larger arteries in the muscular and, to a lesser extent, in the elastic vessels. Though patchy in distribution in any one vessel, in all three, evidence of generalized vascular damage was present. The smaller arteries and capillaries in the various organs were not appreciably affected in any way. Likewise, no definite changes were found in the veins of any of these cases.

Of changes observed in elderly diabetics, the material examined was obtained without exception from the lower limbs of sixteen patients who had suffered from diabetic gangrene necessitating amputation. In consequence, only the findings in the muscular arteries will be considered.

In contradistinction to the previous cases, the changes found were of marked degree, and presented such a variation in appearance as to render the task of correlation extremely difficult. The details of the findings in this series have been considered in detail elsewhere,[14] so that a mere summary will suffice here.

In all these cases obliterative changes in one or more of the main arteries of the limb were present. The mechanism of the production of this occlusion seemed to be somewhat different in different cases. In some aggregations of fat and of cholesterol were responsible. In others intraarterial thrombosis had occurred with subsequent organization of the clot, and yet in others a bland hyaline thickening of the intima had occurred. Lastly, in a few cases a neovascularization, such as that described by Letulle, Labbé and Heitz,[15] had apparently caused a progressive encroachment on the lumen by an organizing connective tissue arising in the intima.

Gross degenerative changes were present in the media in all cases. These varied from fatty degeneration and infiltration to calcification and even ossification. In others, the site of old occlusive damage, a disuse atrophy of the muscular coat was found. In one case small aneurysmal dilatations were present along the course of the posterior tibial artery.

No change was apparent in the adventitia apart from an increase in the number of periarterial arterioles in cases in which the main artery had ceased to function adequately.

Once again the examination of the smaller arterioles and of the capillaries revealed comparatively little alteration in structure. Occasionally signs of endarterial activity in the way of proliferation of endothelium was seen, but in general the healthy appearance of the smaller vascular channels was in striking contrast to the gross changes observed in the main vessels.

An entirely different aspect of the problem of vascular disease arose as a result of the observations of Schmidtmann and Hüttich on animals in 1927.[16] They found that the arteries of cows were inclined to show an acid reaction to appropriate indicators, whilst the arteries of calves showed an alkaline reaction. Two years earlier Peyton Rous published the first of a series of papers on "The Relative Reaction Within Living Mammalian Tissues".[17] He established beyond doubt that the pH values of various animal tissues undergo considerable variation under experimental conditions.

It occurred to the writer that some alteration in the hydrogen ion concentration of the arterial wall might possibly occur in the presence of vascular disease, and an attempt was made to determine this fact in the following way. During the amputation of the leg of a woman, aged sixty-three years, for diabetic gangrene, Mr. Balcombe Quick isolated the anterior tibial artery and directly injected it with 10 cubic centimetres of a 2% solution of phenol red. This dye, which possesses a range of hydrogen ion concentration from 7·6 to 6·8, was selected after experimentation as being the most suitable for the purpose. The artery was then tied off and the amputation proceeded with. Immediately after removal the artery was dissected out under liquid paraffin in order to prevent the access of air. The vessel was found to be grossly diseased, and from the colour changes observed in the wall the hydrogen ion concentration was seen to vary from 6·8 to 7·0. Control experiments upon limbs amputated for traumatic damage in subjects of the same age have established the fact that the normal figure for the arterial wall is approximately that of the blood, namely, 7·4.

Discussion.

In attempting any consideration of vascular disease one is reminded of the words of Boyd: "The subject is one of great difficulty, confusion and obscurity." The causation of arteriosclerosis has been the subject of a vast literature, but the problem remains unsolved. No experimental work has been published on the direct production of vascular disease in the diabetic animal.

In the experimental work upon arterial degeneration the following two methods have been used chiefly: (*a*) The feeding of certain substances to animals over long periods of time. (*b*) The production of direct injury to the vascular system by the injection of bacteria and certain drugs, for example, adrenaline, or by a prolonged strain thrown upon this system by artificial means.

Ignatowski[18] and Saltykow[19] produced intimal lesions in the rabbit by the feeding of abnormal diets over a period of many weeks. Later Steinbiss,[20] Anitschkow,[21] and Chalatow[22] produced similar lesions by dietary measures. The essential feature of all these experiments was the high proportion of animal food which is normally foreign to an herbivorous animal, such as the rabbit.

Ignatowski concluded that the high proportion of animal protein was responsible for the lesions, but Stuckey[23] maintained that the high lipoid content of the diet was the determining factor, and Saltykow[24] in a later paper supported this view. Knack[25] then repeated some of the earlier experiments and showed that rabbits fed upon an ordinary diet containing large amounts of cholesterol did not develop vascular lesions, while rabbits fed upon diets of animal protein showed definite vascular change. He concluded that a simple hypercholesterolæmia did not produce arteriosclerosis. Newburgh and Clarkson[26] have shown recently that rabbits fed on protein diets with very little cholesterol develop vascular changes. The latest work in this field is that of Sansum,[27] who claims that rabbits fed upon acid ash protein develop arterial changes, whilst rabbits fed upon alkaline ash protein do not show any such change.

The vascular damage produced by the injection of bacteria and of adrenaline calls for little comment, as it has not much bearing upon the particular aspect of the subject under discussion.

As to the initial phenomena to be observed in the early stages of vascular degeneration in the human being, Virchow's views are accepted to the present day. He states:

> There occurs a certain loosening of the connective tissue ground substance of the intima which must be attributed in a large measure to the increased imbibition of fluid elements from the passing blood stream and is recognized microscopically by the increased width and homogeneity of the connective tissue spaces.

Aschoff,[28] in elaborating Virchow's opinion, postulates two chief factors in the development of vascular lesions: (i) An abnormal increase in imbibition and a certain slowing of the plasma which normally nourishes the inner layers of the vessel wall. (ii) A sufficient concentration of lipoids, especially cholesterin esters, in the plasma.

From the observations on the vascular lesions of adolescent diabetics, it seems probable that the initial lesions are similar to those described by Virchow.

Considering next the relationship of *diabetes mellitus* in more detail, there is an ever increasing weight of opinion that this disease is not only associated with a disturbance of carbohydrate metabolism, but that other metabolic processes are affected as well.

In many cases of diabetes a marked rise of the fat and lipoid content of the blood occurs, the explanation of which is still a matter of controversy. Joslin[29] favours the view that this increase is due to the inability of the diabetic to metabolize adequately the fats ingested in the food. Rabinowitch[30] has shown that, although no direct relationship exists between the plasma cholesterol and the blood sugar, in the severe cases and in those inadequately controlled the plasma cholesterol is definitely raised. This increase is occasionally marked by the deposition of fats from the circulation, as in *lipæmia retinalis*, or by the deposition of

lipoids, as in *xanthoma diabeticorum*, and in the deposits found at times in the spleen and other parts of the reticulo-endothelium. Aschoff, as already mentioned, believes that a hypercholesterolæmia is necessary for the deposition of lipoids in the intima, and it is possible that such deposition may occur under suitable circumstances in the diabetic. Bloor,[31] on the other hand, states that the deposition of cholesterol and its esters does not necessarily require a high blood cholesterol, but that it depends upon the ability of the blood to keep in solution a substance which is probably in a state of supersaturation.

Another phase of this disturbed lipoid metabolism is seen in the work of Fishberg,[32] who has shown that a definite relationship exists between the colloidal osmotic pressure of the blood and its lipoid content. Rabinowitch,[33] after the study of sera from a group of diabetic patients, suggests that "the sum of all the available data tends to support the view that in diabetes with lipæmia there is constantly exerted in the capillaries a colloidal pressure greater than normal". He suggests that this is a possible factor in the development of arterial degeneration in the diabetic.

Be this as it may, the disturbance of fat and lipoid metabolism would appear to be of some importance in the development of arterial disease in view of the findings described herein and with regard to the accepted views upon the early stages in the development of arterial degeneration.

At this stage the question arises as to whether there is a characteristic diabetic arteritis. The wide variation in the appearances found in the cases already considered does not permit an affirmative reply. The organizing processes in the intima are suggestive of some phases of Buerger's disease. The finding of aneurysmal dilatations is reminiscent of *periarteritis nodosa*, the occurrence of medial calcification resembling Monckeberg's sclerosis. All serve to illustrate the variation in the types of response of the arterial wall to the various factors concerned in the production of this vascular damage. The comparative escape of the smaller vessels and of the capillaries calls for further comment. The absence of any appearances suggesting diffuse hyperplastic sclerosis, as described by Evans[34] and Kernohan,[35] and the absence of any evidence of medial hypertrophy, are of much importance. Herein lies the explanation of the blood pressure findings already considered.

The unusual predilection of arterial disease for the muscular arteries of diabetics is also worthy of comment, as, whatever be the reason for this interesting fact, it explains the unusual sites of the vascular accidents in these patients.

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X RAY DEMONSTRATION OF FRACTURE OF THE NASAL BONES.

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FRACTURE of the nose is a very common injury, generally arising from direct violence. In the routine examination of the head in the postero-anterior and lateral positions, the examination of the nasal bones and the structures in the immediate neighbourhood is most unsatisfactory; in fact, it is

difficult to recognize the nasal bones at all, much less identify any fracture which might exist.

For many years I have been examining the region of the nasal bones by means of dental films, the technique being extremely simple. A film is held by the patient after being placed in position as high up on one side of the nose as possible, being limited only by the medial part of the suparorbital ridge; the exposure is then made perpendicularly to the film through the opposite side of the nose. This process is repeated on the other side. Particular note is taken as to which film is placed on the left side, which on the right.

The illustrations show the frontal nasal and maxillo-nasal sutures very clearly, whilst the lines of fracture are definitely demonstrable and cannot be shown so clearly in the usual skiagrams of the head.

The fact that I have not seen this simple technique described and that I believe it is not generally known, provides the excuse for describing this procedure.

HÆMATURIA AND ITS INVESTIGATION.[1]

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The appearance of blood in the urine, furnishing as it does positive evidence of organic disease, calls for an immediate and thorough examination of the patient, which cannot be regarded as complete until the pathological foundation of the bleeding is established beyond all reasonable doubt. While in many instances this is impossible without a complete urological investigation, in a large number a very fair conception of the nature of the lesion can be gained by clinical methods alone, and frequently, provided the clinical examination be complete, nothing further may be necessary to establish the diagnosis.

Ætiology.

The causes of hæmaturia can be most conveniently divided into four main groups. These are trauma, general diseases, developmental abnormalities and local disease.

Trauma.

Hæmaturia of traumatic origin may originate anywhere in the urinary tract, but most commonly arises from the kidney, following violence applied to the loin, or urethra, as from a crushing injury, or occasionally associated with the passage of a foreign body. Blood resulting from mild instrumental trauma is very common in urine obtained by ureteral catheterization, while the passage of a catheter or other instrument into an inflamed bladder or the dilatation of a urethral stricture is also very prone to cause some bleeding.

General Diseases.

Hæmaturia may arise in the course of a general systemic disease, such as the malignant fevers, hæmophilia, scurvy or purpura, or it may be due to a renal infarct occurring in the course of pyæmia or endocarditis.

In these two groups the cause of the hæmaturia is usually fairly obvious, and for the purposes of this paper they may be dismissed.

Developmental Abnormalities.

The only cause of hæmaturia falling under the heading of developmental abnormalities is polycystic disease of the kidneys. In this condition, while the renal parenchyma is compressed by the cysts, the vessels are more resistant and stand out as linear projections from the cyst walls. As a result of excessive tension, sometimes precipitated by quite slight trauma, a vessel may rupture into a cyst and the cyst in turn may rupture into the pelvis, causing hæmaturia which may even endanger life.

Local Disease.

Hæmorrhage from the Kidney and Renal Pelvis.

Hæmorrhage may arise from the presence of a non-surgical lesion, such as acute nephritis, either infective or due to poisons, acute hæmorrhagic or focal nephritis, syphilitic nephritis, arteriosclerosis or chronic nephritis, and in all these conditions it may be quite profuse. More commonly, however the lesion is surgical. Of non-infective lesions the commonest are calculus and neoplasm. Hydronephrosis not due to calculus is an unusual cause, but occasionally gives rise to severe bleeding. Colic due to any cause may be responsible, the bleeding following bruising of the mucosa and rupture of small vessels, while less common causes are angiomata and nævi of the renal pelvis and sudden emptying of a chronically distended bladder. Again, the lesion may be infective, either specific, such as tuberculosis (though renal bleeding here is the exception), or a non-specific pyelonephritis, whether primary or complicating calculus, hydronephrosis or tuberculosis, or secondary to a vesical infection. At other times the lesion may be parasitic, due to hydatid disease, or very rarely of actinomycotic origin.

Hæmorrhage from the Ureter.

Ureteral bleeding may accompany colic due to any cause, calculus, oxaluria, or passage of caseous *débris* or hydatid daughter cysts, or the colic which is frequently seen without obvious pathological basis. On rare occasions it may be due to ureteral neoplasm, or *ureteritis cystica*, and is sometimes encountered in acute appendicitis, when the appendix overlies the ureter, and in parametritis.

Hæmorrhage from the Bladder.

Vesical hæmorrhage may be due to various non-infective lesions, of which the most important by far is neoplasm. It may be associated with calculus,

[1] Read at the second annual reunion of the Residents' and Ex-Residents' Association, Royal Prince Alfred Hospital, Sydney, October, 1931.

and is very common in the presence of hypertrophied prostate, but, strangely enough, rather the exception in prostatic carcinoma. When accompanying prostatic obstruction it is due either to congestion of the vesical neck and prostatic urethra, or to the rupture of dilated venules on the vesical aspect of the enlarged gland. Vesical neck congestion, or *cystitis colli*, and trigonitis are a frequent cause of hæmaturia in women, and may be secondary to acute gonorrhœa, to inflammatory disease of the uterine adnexa, or, most common of all, to the senile urethral stricture formation often met with after the menopause. In men the condition is usually secondary to prostatitis. Another, though rare, source of hæmaturia is a split in the mucosa of a bladder which is the seat of submucous or interstitial cystitis. Again, the hæmaturia may be of infective origin. A non-specific infection may be present, either a primary acute cystitis or a chronic cystitis secondary to calculus, neoplasm, vesical neck or urethral obstruction, to renal or prostatic infection or to a foreign body. The infection again may be specific, due to tuberculosis, or parasitic, due to bilharzial infestation. On rare occasions a tertiary syphilitic lesion may be present.

Hæmorrhage from the Urethra.

Bleeding from the posterior urethra may accompany prostatitis and posterior urethritis, which may be either of gonococcal origin or non-specific, usually staphylococcal. Bleeding from the anterior urethra is rare. It may be due to a calculus or neoplasm, and is stated to occur sometimes in the presence of acute gonorrhœa. In the female it may arise from a caruncle.

No mention has been made of essential hæmaturia. This condition can be regarded only as a last and unsatisfactory diagnostic refuge when all else fails. Many cases so classified are probably due to nephritis, others to small angiomata or nævi of the renal pelvis, while others again have been found by pyeloscopy to be apparently due to papillary congestion secondary to spasm of the ring muscles surrounding the bases of the renal papillæ.

Clinical Examination of the Patient.

Needless to say, the history must be complete. The patient should be questioned regarding previous attacks of hæmaturia, whether the blood is bright or dark, the extent to which the urine is discoloured, and whether or not the blood is evenly mixed. Associated symptoms, such as renal pain and disturbances of micturition, should always be inquired into, as they may not be very marked, and the patient, with his attention concentrated on the hæmaturia, may not mention their presence. The same statement applies to general symptoms, such as malaise, rigors or loss of weight. Inquiry must be made into previous illnesses, especially the infectious fevers, as possible antecedents of nephritis. If a history of gonorrhœa is obtained, the type of treatment received and its duration should be ascertained, as affording information as to the likelihood that a gonococcal lesion of the prostate and vesicles may still persist.

Physical examination must be equally complete. It may seem unnecessary to stress the need for an abdominal examination, but cases of painless hæmaturia have been known in which it has been omitted, and a diagnosis of polycystic kidney consequently missed. Not only in men at the prostatic age, but at all ages, a rectal examination should never be omitted, unless, of course, the cause is obviously renal, and if the prostate is not hypertrophied its secretion should be expressed and examined microscopically. Similarly, in women a vaginal examination should always be carried out in doubtful cases and the condition of the uterus and adnexa determined. The external urethral orifice also should be examined for evidence of caruncle or for the prolapsed and engorged mucosa which is usually the pathological foundation of a caruncle and not an entity in itself, but evidence of trouble higher in the urethra in the form of a stricture. Examination of the cardio-vascular system in doubtful cases, with blood pressure determination, may reveal evidence of chronic nephritis, and in children the throat should always be examined for focal infection, the origin of a possible focal nephritis.

Next, as regards the urine, if bleeding is still active when the patient is seen, the three-glass test gives some limited information regarding its source. If blood drips from the urethra independently of micturition and the first glass is stained while the others are comparatively clear, the lesion is below the external sphincter, in the anterior urethra. If there is no bleeding between voidings, the blood must come from above the external sphincter. If the source is in the posterior urethra, the first glass will be stained, and with only moderate bleeding, the second and third glasses may be clear, or the third glass may be stained, owing to blood being squeezed out by the sphincters at the end of micturition. With more profuse bleeding the blood finds its way back into the bladder and all three glasses become stained. Blood coming from above the vesical sphincter, whatever its origin, is usually well mixed with the urine, but occasionally terminal hæmaturia, with blood in the third glass only, may be caused by the bladder contracting on a neoplasm at the end of micturition, or by a frond of villous growth being caught by the internal sphincter. Though suggestive of neoplasm, it is not pathognomonic, as it may occur with a very engorged bladder wall, arising from any cause, and also occurs in bilharziasis. When all three glasses are stained, the only diagnostic points on which reliance can be placed are the presence of ureteral casts of blood clot, indicating supravesical bleeding, and blood casts, which are positive evidence of bleeding from the renal parenchyma. Bright red blood almost always arises from the bladder or posterior urethra, while dark blood may be equally well coming from the upper or lower tract.

Testing for albumin does not usually give very satisfactory information, as it is always present. In an acute nephritis the amounts of albumin and blood are usually quite obviously out of proportion, while with a less noticeable difference it is possible to estimate any discrepancy in their relative amounts by counting the red cells present in one cubic millimetre of urine, calculating thence the amount of albumin which should be present from the fact that 100 cubic centimetres of blood contain about 7·5 grammes, and estimating the amount that actually is present with an Esbach's albuminometer. If pus is present in addition, however, this procedure is quite useless.

Even though the presence of blood is quite obvious to the naked eye, microscopical examination of the urine should never be omitted. In the first place, it is the only means of insuring that the very occasional case of hæmatoporphyrinuria is not missed, and secondly, it is most important to discover whether or not other elements than blood are present. Casts should be looked for, while if pus is present, a search for organisms should be made. Bacilli, if present, are quite easily recognized under the high power of the microscope as motile rods. Very occasionally the examination is rewarded by the unexpected discovery of hydatid hooklets.

Clinical Diagnosis.

Hæmaturia Associated with Renal Symptoms.

When pus is absent, the commonest condition met with under this heading is recent severe colic, with hæmaturia, often, but not necessarily, due to calculus. If the skiagram reveals no abnormality and the condition clears up, nothing further need be done save keep the patient under observation for any sign of recurrence, when further investigation becomes necessary. Recurring attacks of colic and hæmaturia, especially if brought on by exercise, are very typical of renal calculus, while if the history is one of slight renal pain, with comparatively marked hæmaturia, suspicion of neoplasm should arise.

When pus is also present, the history is usually recent and the cause generally an acute pyelonephritis, further evidence of which will be afforded by a history of one or more rigors, the presence of pyrexia and lumbar tenderness, and the rapid improvement in the patient's condition under medical treatment. If this improvement does not take place, cystoscopy to determine the factor which is preventing spontaneous recovery should not be too long deferred. Chronic infective renal lesions, which are practically always secondary to obstructive lesions, are not usually accompanied by much bleeding unless there is a good deal of associated cystitis. If there is much pyuria, and the condition is of fairly long standing, a disorganized kidney requiring removal is usually present.

Hæmaturia Associated with Vesical Symptoms.

A mild degree of frequency of micturition and scalding very frequently accompanies hæmaturia of any degree or renal pain of any severity, and for the purpose of locating the primary pathological lesion is of little diagnostic value. The presence, however, of marked disturbances of micturition as an integral part of the clinical syndrome, when taken in conjunction with the urinary findings, gives very valuable information. With a history of obvious vesical disturbance, moreover, the presence of renal symptoms should always be closely inquired into, since with a severe hæmorrhagic cystitis it often happens that a mild degree of unilateral renal pain may be the only clinical indication of the presence of a disorganized kidney which is the primary cause of all the trouble and whose removal is necessary before the cystitis will clear up.

Hæmaturia Associated with Difficulty of Micturition.

In men the commonest cause of difficulty of micturition is prostatic hypertrophy, which will usually be evident on rectal examination. Prostatic hypertrophy and vesical neoplasm, however, occur not uncommonly in association, so that the discovery of an enlarged prostate does not complete the investigation. Cystoscopy should be carried out in addition. When rectal examination reveals no abnormality, the hæmaturia may arise from congestion or infection secondary to an enlarged prostatic middle lobe or a prostatic bar. In other cases it may be due to urethral stricture, when the hæmaturia is generally due to associated prostatitis and posterior urethritis. In women difficulty in micturition is most often associated with senile urethral stricture formation, or occasionally to a marked degree of cystocele. Occasionally hæmaturia may arise as a result of secondary infection of a bladder whose innervation and emptying capacity are interfered with by central nervous disease.

Hæmaturia Associated with Frequent and Grossly Painful Micturition.

Hæmaturia associated with frequent and grossly painful micturition is usually due either to acute or to tuberculous cystitis. Acute cystitis will be recognized by its sudden onset, recent history, the presence of pyuria with motile organisms, the easy culture of pathogenic organisms on ordinary media, and by the rapid improvement in the patient's condition that takes place under medical treatment.

In tuberculous cystitis the more insidious onset, the longer history, the presence of pyuria without visible organisms and sterility of the urine on incubation on ordinary media, unless secondary infection has occurred, and the resistance to medical treatment, will suggest the actual diagnosis and indicate the need for further investigation to determine which kidney is the seat of the primary lesion.

Other causes are not common. Occasionally bleeding occurs from a Hunner's ulcer. This condition fortunately is rare. The ætiology is unknown, but the entire bladder wall becomes involved in a process of fibrosis, with markedly diminished bladder capacity, gross dysuria and frequency of micturition, and a urine free from any abnormal

constituents. Occasionally a little distension in excess of the usual tolerance of such a bladder causes a linear split in the mucosa, the so-called elusive ulcer, from which some bleeding may arise. The hæmaturia due to bilharzial infestation also is frequently accompanied by severe perineal pain. Occasionally a non-specific chronic cystitis secondary to any cause may lead to hæmaturia accompanied by grossly painful micturition.

Hæmaturia Associated with Frequency of Micturition, but without Severe Pain.

With Pyuria. The presence of pus naturally indicates an inflammatory origin of the bleeding. With a recent history the condition is most probably one of simple acute cystitis, which will be determined by the same criteria as already mentioned. Especially in women, however, when a catheter specimen has not been obtained, it is most important to make sure that the patient is not suffering from acute gonorrhœa, and suspicion of this should always arise if no organisms can be found in the fresh specimen of urine.

When the history is of longer standing or when medical treatment fails to relieve an acute cystitis, the problem resolves itself into the determination of the primary lesion which is responsible for the cystitis and is preventing it from resolving. When this is found and treated the cystitis will heal and the hæmaturia disappear. As before, a urine sterile on attempted culture, or absence of visible organisms will suggest the presence of tuberculous disease, though the same finding is occasionally obtained in chronic infections complicating calculus. If motile organisms are present, the primary lesion may be an obstructive one, such as prostatic hypertrophy or carcinoma, prostatic bar or urethral stricture with or without calculus, all of which may prevent a vesical infection from clearing up without causing very much actual difficulty in micturition; or a non-obstructive lesion, such as vesical neoplasm, prostatitis or upper tract infection.

Without Pyuria. The most important potential cause under this heading is vesical neoplasm, the presence or absence of which requires to be definitely established. Usually, when patients with this disability first seek medical advice, there is very little disturbance in micturition, but not necessarily so. Again, vesical calculus may be present, usually secondary to vesical neck obstruction. Vesical neck congestion and trigonitis as causes of frequency of micturition and hæmaturia are very common, especially in women, probably the commonest cause in this category. In the male they may be secondary to vesical neck obstruction due to any cause, or to prostatitis, which may be gonococcal in nature, either recently acquired or of long standing, or to a non-specific prostatic infection by the common pyogenic organisms. In the female the condition is most commonly associated with senile urethral stricture, and may occur in the presence of inflammatory disease of the uterine adnexa, either gonococcal or otherwise, or of uterine displacements.

Symptomless Hæmaturia.

With Pyuria. This is not common. It is stated that a brisk hæmaturia may be the first indication of a tuberculous process in the kidney, in the same way as a hæmoptysis may be the initial symptom in pulmonary tuberculosis. If this occurs, it is very rare. A commoner condition is a moderate amount of hæmaturia, with much pus and *débris,* but with little disturbance of the act of micturition, which is very characteristic of advanced carcinoma of the bladder.

Without Pyuria. These cases fall into two groups. In one group a complete examination may either disclose or suggest the source of the bleeding, so that no avenue should be left unexplored. The important point to remember is that symptomless hæmaturia may be due to practically any lesion anywhere in the urinary tract. Abdominal examination may disclose a renal tumour or two large polycystic kidneys which have given no other indication of their presence. Hæmaturia in fact is the symptom which most often leads patients suffering from polycystic disease to seek medical advice. Valuable findings again may be obtained by rectal or vaginal examination or examination of the expressed prostatic secretion. The possibility of nephritis should never be forgotten, in children especially. In a child the diagnosis of whose condition is at first sight quite obscure, the discovery of casts, the presence of rather more albuminuria than would be expected from the amount of blood present, and a pair of chronically inflamed tonsils will practically establish the diagnosis of acute hæmorrhagic nephritis.

Very frequently, however, the examination reveals no abnormality and not the slightest indication is present as to the source of the bleeding. Such complete absence of associated signs and symptoms should give rise to immediate suspicion of vesical neoplasm and cystoscopy is imperative; it is none the less necessary because the hæmaturia may be slight or transient, or because the patient may be regarded as too young. Papilloma of the bladder may occur at any age and is always potentially malignant, and after the first attack of hæmaturia it may give no further indication of its presence for months or even years. Treatment by cystoscopic diathermy of early papilloma is so satisfactory and the eradication of advanced growths so difficult, that the patient should have the benefit of a cystoscopic investigation as soon as possible.

Cystoscopic Investigation.

As the possibility of neoplasm is always present, every part of the bladder wall must be inspected. That is to say, it is essential to use the indirect cystoscope. Failure to find a neoplasm with a direct cystoscope in the limited area available for inspection is of no value whatever as an indication of its absence.

The findings with the indirect cystoscope, however, are conclusive and, of course, are of no less value if no abnormality is seen; if the hæmaturia has ceased the source of the bleeding may not be

ILLUSTRATIONS TO THE ARTICLE BY DR. H. FLECKER.

Skiagrams showing fractures of the nasal bones.

ILLUSTRATION TO THE ARTICLE BY DR. C. J. OFFICER BROWN.

Skiagram taken with the patient standing. Note the layer of gas separating the right dome of the diaphragm from the upper surface of the liver.

ILLUSTRATIONS TO THE ARTICLE BY DR. E. W. FAIRFAX AND DR. OLIVER LATHAM.

FIGURE I.
Paraffin section, hæmatoxylin and eosin stain, pia from lower lumbar region. The thickened pia, with its mononuclear infiltration, is well seen. (200 times natural size.)

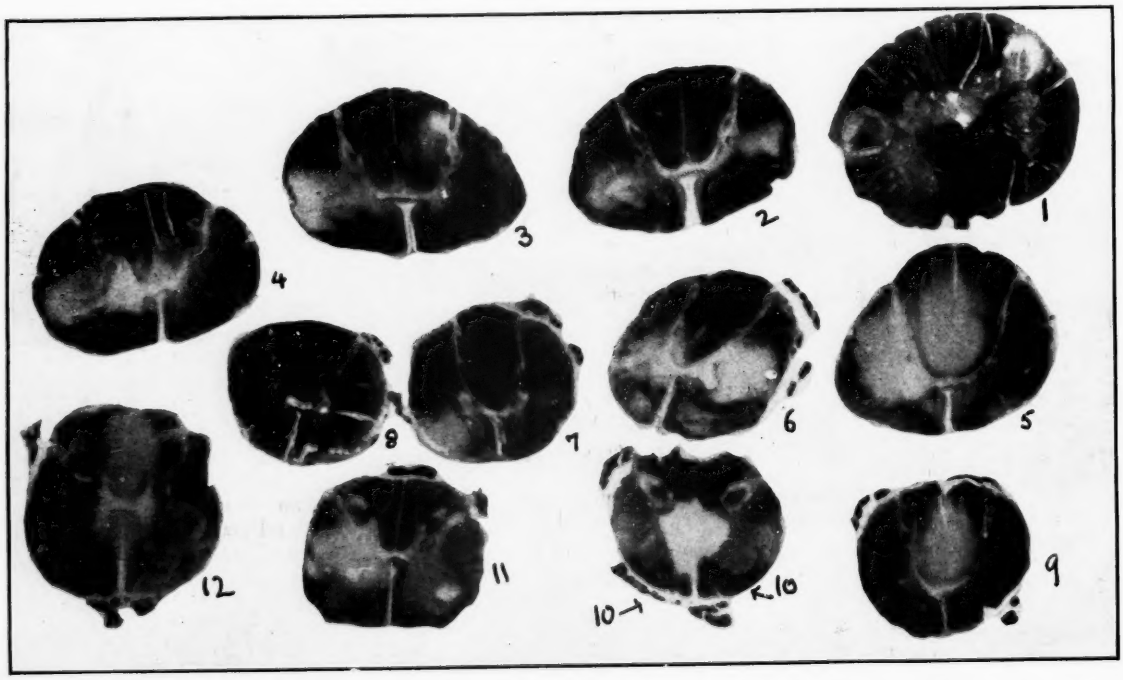

FIGURE II.
Sections from lower medulla and eleven levels of cervical, dorsal and lumbar regions in celloidin, Weigert's stain. The peculiar distribution of the myelitis or sclerosis with Section 8 midway between them, almost normal, can be noted. Also Section 10, where the gliosis is most central and more than elsewhere demyelinates the grey matter. (Twice natural size.)

obvious at all. For this reason cystoscopy should, if at all possible, be carried out while the bleeding is in progress. This makes it much more difficult technically, but only if there is free bleeding of vesical origin will the field be too obscured to make a definite diagnosis, and in this event the patient may be reexamined after the bleeding has ceased, in the certain expectation that the source of the hæmaturia will be visible, while if intractable bleeding persists suprapubic cystotomy can be performed in full confidence that the lesion will be exposed. With bleeding of renal origin it is always possible to carry out a satisfactory examination, and inspection of the ureteral effluxes will demonstrate which kidney is the offender. This method is much to be preferred to that of examining specimens of urine obtained by ureteral catheterization. As previously mentioned, such specimens often contain traumatic blood and are sometimes quite grossly stained. In other instances blood can be seen leaking into the bladder between the cystoscope and the internal sphincter, obviously coming from the posterior urethra.

If, however, the patient is examined after the hæmaturia has ceased, and there is no lesion in the bladder, the problem is more difficult. Tests of renal efficiency may provide useful information. An X ray picture should always be taken, as painless hæmaturia may be, and often is, the first symptom of unilateral or bilateral staghorn calculi, and sometimes even of the more common rounded or irregular variety of stone. If the skiagram shows no abnormality, bilateral pyelography can be carried out, but is not a very desirable proceeding. Better procedures are intravenous pyelography, or an estimation of differential renal function with indigocarmine or phenolsulphonephthalein, when an impaired excretion on one side will frequently denote the presence there of the lesion sought for, the nature of which will be disclosed by pyelography, while impaired excretion on both sides, in the absence of a bilateral surgical lesion, can be due only to chronic nephritis. If there are still no signs of abnormality, the patient should be instructed to report for investigation if and when the hæmaturia reappears.

Conclusions.

Several points emerge from this paper which may with advantage be stressed once more.

1. The majority of cases of hæmaturia require cystoscopic investigation for their complete diagnosis.

2. All patients with symptomless hæmaturia, without obvious basis, must be regarded as suffering from neoplasm until the contrary is proved.

3. Though what appears to be the primary cause of an infective hæmaturia may be suggested by clinical examination, it is not safe to regard it as such unless both the hæmaturia and pyuria clear up completely under treatment.

4. Medical treatment, if not definitely influencing for the better a case of cystitis and hæmaturia, should not be persisted with for too long.

5. The possibility of acute gonorrhœa should always be eliminated before hæmaturia is ascribed to acute cystitis.

6. Painless hæmaturia is often the first symptom of polycystic disease and of staghorn calculi.

Reports of Cases.

ACUTE DISSEMINATED SCLEROSIS ACCOMPANIED BY POLIO-ENCEPHALITIS.

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Cases of disseminated sclerosis running an acute course, at least ending in death within a few weeks of the patient's seeking medical aid, are sufficiently rare to merit report. This very acuteness masks the real pathological basis of the condition, and consequently the disease is easily missed and other neurological diagnoses are made.

In recent years many confusing neurological conditions have turned out to have the pathological basis of an encephalitis and this disease has come to be included among the diagnoses.

Commonly speaking, multiple neuritis and encephalitis have a pathology reasonably distinct from each other. However, under the term encephalomyelitis have been described many interesting cases with some of the pathological features of both, and, of course, in both diseases clinical phenomena are proverbially fleeting, capable of change from day to day.

In the case we propose to describe the pathological changes of both *encephalitis lethargica* and disseminated sclerosis seemed developed in quite a specific manner, and the patient died within twelve days of the development of serious symptoms.

Case History (E. W. Fairfax).

Mrs. R., pregnant four months, complained of "weakness of the legs" and "tiredness of the nerves" during the last six days. Quite well up to six days ago, she suddenly lost the use of her legs. She had been very thirsty. (At a later date, however, it was ascertained from relatives that she had been gradually getting weak in both arms and legs and was becoming more and more listless and used to wander in thought and expressions during the previous two years, though apparently not to such a degree as to warrant medical aid till the present time.)

There were three healthy children, the eldest being four years of age, and there were no histories of miscarriages. Before and after the patient came under observation there was no evidence of cough, nausea, vomiting or dysuria. However, her bowels were costive, and on examination her urine contained some glucose and a trace of albumin. Later examination revealed a normal blood glucose tolerance curve.

On admission the patient had a bronzed complexion and sunburnt cheeks. Her expression was languid. Her temperature was normal (later becoming elevated), the pulse 120 a minute and the respirations 24. Her abdomen was neither tender nor rigid and the height of the *fundus uteri* agreed with a four months pregnancy.

There was marked muscular weakness in both legs, without any wasting or tenderness in the muscles, and a provisional diagnosis of diabetic peripheral neuritis was made. Her diet was arranged in accordance with these findings.

Two days later there was urinary retention with overflow incontinence.

Examination of the nervous system showed that the cranial nerves revealed no detectable abnormality. There

was marked loss of power in both extremities, and the hand grips were weak on both sides. Sensation to light touch and pain was much impaired on the left side from the middle of the thigh downwards. Sense of position was poor in both legs and arms.

Knee jerks were both present, with the left more active. Biceps jerk was bilaterally present. The patient's mental condition seemed definitely impaired.

Two days later astereognosis was marked in the upper limbs, mental confusion persisting.

Four days later the diagnosis seemed to lie between: (i) disseminated sclerosis, (ii) syringomyelia, and (iii) encephalitis. Diabetes was ultimately excluded, since the sugar disappeared from the urine on simple treatment, and the glucose tolerance test gave a normal result. The temperature showed an almost daily rise up to and occasionally over 37·8° C. (100° F.). The patient became weaker and ultimately died twelve days after admission.

Laboratory Reports.—The following reports were received from the laboratory. The Bordet-Wassermann serum reaction was normal. The blood sugar content was normal. The urine at first contained a little sugar and albumin which cleared up. There were no casts, pus *et cetera*, and on incubation the urine proved sterile.

Pathological Report (Oliver Latham).

The brain and cord, preserved in formalin, of a female aged thirty-five, was received for examination and report. The accompanying report stated that the symptoms during life suggested: (i) disseminated sclerosis, (ii) syringomyelia, (iii) encephalitis. The final illness apparently lasted under twelve days.

On examination the pia-arachnoid over both hemispheres and to a less extent elsewhere appeared suffused with blood which, however, did not exceed three millimetres (one-eighth of an inch) in thickness. The pia was thickened.

The kidneys showed acute catarrhal nephritis, the liver acute congestion and cloudy swelling. The spleen was enlarged and markedly congested. In the lung pneumonic consolidation in various stages was found.

The brain and spinal cord were sectioned and no further abnormalities were noted, save that the spinal cord would not stand handling (nutritional atrophy), and twelve additional levels had to be put through celloidin before treatment.

Technique.—Pieces of cord from some sixteen levels from medulla to lumbar region were put through paraffin for hæmatoxylin and eosin stains, neutral red and Nissl's stain, while frozen and celloidin sections were mordanted for Weigert-Pal myelin sheath stains. Van Gieson's stain was used for neuroglia, Cajal's method for axis cylinders, Marchi's method for degenerating myelin. Sections from the brain for paraffin and hæmatoxylin, eosin and Van Gieson's and Nissl's stain were taken of areas from the frontal, Rolandic, occipital, hippocampal, optic thalamus, lenticular nucleus, *substantia nigra*, pons, medulla and cerebellum.

Results of Pathological Examination.

It was soon evident that in the cord two sets of phenomena were present. Nearly every level showed some myelin loss, ranging from a few fibres to half the transverse area of the cord, and these degenerated areas at one level had no anatomical relation to those of another level, that is, absence of systemic affection. This showed the lesion to be of the nature of disseminated sclerosis or encephalomyelitis. The large patch of area affected in any one section and the absence of smaller areas around blood vessels seemed to rule out encephalomyelitis, and the intense network of fine gliosis, apparently having its origin in the grey matter, was typical of disseminated sclerosis, although present in some other states. In some areas, especially the lower cervical (Figure III), the myelin destruction was particularly acute and recent, with *Gitterzellen* along the blood vessels carrying off myelin droplets. Even here the delicate gliosis was very evident, as brought out by Van Gieson's stain on tissue put through the mordants and treated with permanganate of potash and oxalic acid. Again, the extremely local nature of these sclerotic areas, even though superficially, where acute, resembling a true myelitis, argued an escape of most of the axis cylinders, and this was proved by their showing up mostly undamaged by Cajal's silver axis cylinder stain. Some of the horn cells also were damaged in different levels. It remains to be added that while extensive sclerotic lesions were noted both in the cervical and lumbar regions and part of the dorsal cord, yet one level of dorsal cord is shown almost intact. So far this cord resembled one handed in, curiously enough also by Dr. Fairfax, in 1915,[a] a case of acute disseminated sclerosis in which the diagnosis of acute transverse myelitis was partly justified because one level of cord presented a picture of almost complete transverse softening, but in which also the gliosis was extreme and most of the axis cylinders intact. In the present case no macroscopical areas of sclerosis were noted from the lower medulla upwards, although microscopical areas of sclerosis could be seen in the brain stem as far as the red nucleus, but with little destruction of myelin.

Numerous glia cells, both very large and small, were obtained in both white and grey matter where sclerosed, but especially the grey matter; again, while some loss of myelin from the fine fibres of the grey matter was common, it was extreme in one level of the lumbar region (Figure II, number 10) and the distinct proliferation of ependymal glia so noticeable in our 1915 case and which is supposed to cause the earliest signs, for example, the emotional disturbances of English authorities, was not such a feature of this case.

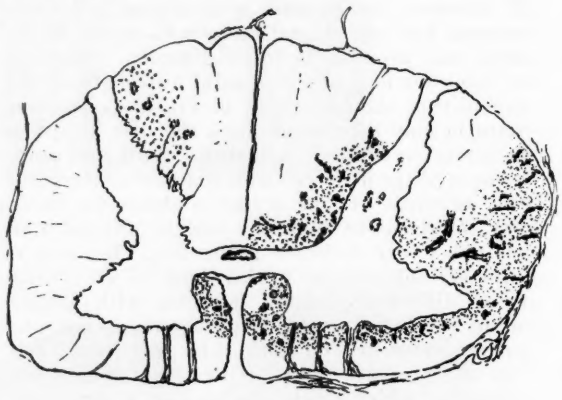

Figure III.

Frozen section from cervical region stained by Marchi's method shows the degenerating myelinated fibres (black) together with the "fat-granule-cells" in the perivascular spaces. Although the grey matter is not shown involved by this process, yet van Gieson's stain shows practically all the involved areas in the section to be joined by the fine gliosis within which are many glia cells. (Ten times natural size.)

The Evidences of a Polio-Encephalitis Reaction.

Side by side with these eminently typical lesions of disseminated sclerosis was an entirely different picture, not previously noted by me in all the cases of disseminated sclerosis I have examined pathologically. However, I recently noted some of them in a case of encephalomyelitis in Sydney. This picture was one commonly seen in brains and cords from cases of encephalitis, from whatever cause. Especially when the encephalitis is associated with damage to the vascular system and leading to widespread perivascular (and sometimes macroscopical) hæmorrhage. This picture includes thickening and dilatation and infiltration with mononuclear cells and extravasated blood cells of the pia and fibro-hyaline degeneration of its blood vessels. Collections of surface endothelial cells account for the pial opacity. I have already noted that naked eye subpial hæmorrhages were present. These vascular and pial conditions pervaded the whole central nervous system. For instance, the pia was morbidly thickened over the Rolandic area and unduly attached to the underlying

cortex. Furthermore, in the brain stem, optic thalamus and red nucleus, besides lymphorrhages and perivascular hæmorrhages, little areas of sclerosis (Wilson's inflammatory foci) might be seen and associated with widespread degeneration of nerve cells. Thus some of the larger cells in the red nucleus have become calcareous, and many have obviously degenerated in the medulla, in the olives and, curiously enough, in the dentate nucleus of the cerebellum, while Purkinje's cells escape. The degeneration belongs to the type called axonal, the cell losing its shape and nucleus and sometimes being indicated only by a mass of pigment. At the same time there is evident such excess of interstitial reaction as to indicate a sclerosing process. These conditions also hold for the spinal cord.

Discussion.

To recapitulate, we have distributed in patches throughout the cord the typical demyelination and glial reaction of disseminated sclerosis and at the same time a non-pus forming inflammatory reaction or at least vascular damage with hæmorrhage and lymphorrhage and fibrosis with nerve cell destruction throughout the central nervous system. To which of these lesions are we to attribute the patient's symptoms? Emotional phenomena are not uncommon in disseminated sclerosis, but rarely are mental phenomena obvious, except perhaps in these very acute cases. At the same time the neurone destruction and the meningeal reaction might cause the mental confusion and leg weakness. Probably both lesions can be accused, but we cannot apportion the guilt.

The Bordet-Wassermann test gave no reaction and, while the kidneys showed a little acute nephritis, the albuminuria and glycosuria cleared up with simple treatment.

In an article by Barré in the *Revue Neurologique* for March, 1930, a case of disseminated arachnoiditis is described as simulating multiple sclerosis.

Differential Diagnosis.

Since some may well dispute this diagnosis and suggest the case to be one of encephalomyelitis, a condition which has been well reviewed by Kinnear Wilson among others lately, we give the following discussion by rival schools.

Many authorities have noted that, as in this case, the apparent acute onset has been preceded by some weakness or ataxia; thus Oppenheim believed such symptoms ended in typical disseminated sclerosis, which might also follow or develop from a myelo-encephalitis due to an infectious disease. R. T. Williamson, discussing the rapid cases in which death occurred in from three to six months, says of their pathology not only that the axis cylinders persist, but that both the typical scleroses and areas more resembling, microscopically and macroscopically, ordinary softenings, occur together in the same person.

In "Collected Abstracts, Multiple Scleroses"[5] it is maintained that "two main groups are mainly definable" and that for some of the others "an infectious ætiology is justifiable". They would admit a special pathology for multiple sclerosis, namely, an encephalomyelitis distributed in spots, in which the myelin goes, the axis cylinders become deformed, disorientated with glia formation, secondary degeneration connecting the lesions being absent, and the lesions are non-necrosing.

Kinnear Wilson,[7] discussing encephalitis and the specific fevers and the various parenchymatous (ectodermal) changes, and again the exudative, infiltrative and proliferative reactions on the part of the mesoderm (vascular and connective tissues) occurring therein, points out that no specificity attaches to their ætiology. Further on he writes that acute hæmorrhagic polioencephalitis (Wernicke) and acute disseminated encephalomyelitis offer suggestive links with disseminated sclerosis.

Jelliffe and White[10] write:

> The problem narrows itself down to the question whether there exists a multiple sclerosis due to a fairly definite pathological process and other clinical types resembling the former so clearly as to be clinically indistinguishable, but in which one finds multiple tumours, multiple endarteritic lesions (syphilitic, arteriosclerotic), multiple encephalomyelitic inflammatory processes *et cetera*.

These authors continue:

> In rare instances the disorder (disseminated sclerosis) is characterized by running a very rapid course . . . the acute disseminated sclerosis of Marburg. Of late years there has been a tendency to relegate this acute syndrome to the encephalalitides.

We keenly feel the criticisms of those who would place this case of ours in this latter group and will therefore allude to other factors in the pathology of these conditions considered by these authors.

> The sclerosis seems to have a special fondness for places rich in neuroglia . . . though central gliosis is rare . . . these places include near the ventricles, the olives and the *dentate nucleus in the cerebellum*. Small holes may be found in the sclerosed plaques, and in the latter many axis cylinders persist. The more intense the lesion, naturally the fewer neurones will permanently persist, and also different stages of the process will be found in different, even the same lesion. The immense mass of glia fibrils is an entirely new growth . . . apparently not to take the place of the destroyed nervous tissue, but a pure addition product . . . a proliferative inflammation.

They recognize the difficulty confronting those who would argue whether it is a non-purulent encephalomyelitis or a non-inflammatory destruction due to non-infective causes. Various infective agencies have been put forward even quite recently, but await confirmation.[2,9]

Dawson's researches[3] seem to show that there is: (i) A commencing degeneration of myelin sheath and a simultaneous reaction of the glia in the immediate adjacent perivascular tissue, (ii) an increasing glia-cell proliferation and a commencing fat-granule-cell formation, (iii) the stage of so-called fat-granule-cell myelitis, (iv) a commencing glia fibril formation, (v) an advancing, and (vi) a complete sclerosis. Dawson concludes that the underlying process is a subacute disseminated encephalomyelitis which terminated in areas of actual and complete sclerosis. Remissions would then be due to a subsidence and a gradual clearing of the cell exudate and a sclerosing of the tissue with retention of axis cylinders. However, this author fails to show a real inflammatory reaction, and Hassin supports these findings.

The present writers would point out that the distinction between, on the one hand, a patch of ordinary myelitis (that is, one which would be followed in time, if the patient lived long enough, by an ascending and descending secondary degeneration), and on the other hand, a widespread area involving perhaps the whole thickness of the cord of this stage of fat-granule-cell myelitis, is not easy, unless we are allowed to stress: (i) the persistence of so many axis cylinders and (ii) the presence of this remarkably fine gliosis in "disseminated sclerosis". The microscopical appearance of the patches in a case considered by us to be encephalomyelitis differs from this picture. Again, the very acute case of disseminated sclerosis the late Dr. Froude Flashman described with us in 1915 showed extreme fat-granule-cell stage with the least damage to axis cylinders and practically no encephalomyelitis. As that case also exhibited some primitive features in the central canal of the spinal cord, we suggested a developmental or abiotrophic ætiology.

Summary.

1. A case diagnosed as acute myelitis or encephalomyelitis running an exceptionally rapid course has been presented.

2. Microscopical findings revealing the presence of the typical lesions of disseminated sclerosis together with the common signs of encephalitic reactions in both brain, stem and cord have been detailed.

3. Discussions from the literature on these two pathological reactions have been included, and our reasons for the diagnosis of disseminated sclerosis rather than encephalomyelitis set forth.

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DIAGNOSIS OF PERFORATED ULCER BY X RAYS.

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A.O., A MALE, aged thirty, was admitted to the Alfred Hospital at 12.10 a.m. on January 4, 1930, with the following history:

At 7 p.m. on the evening of January 3, 1930, having unloaded some barrels from a motor lorry, he was standing talking when he suddenly felt a severe epigastric pain and fell down in agony. Seen by a doctor fifteen minutes later, he complained of intense generalized abdominal pain, worse in the mid-epigastrium, and of pain on top of the left shoulder; he was ashen and sweaty in appearance, with cold, clammy extremities. His temperature and pulse were subnormal and his abdomen as "hard as a board".

He did not vomit and would admit to no previous history of abdominal pain or indigestion, except that three years before he had "strained his stomach". A diagnosis of perforated gastric ulcer was made and the patient was sent to hospital.

On arrival in hospital five hours after his catastrophe, his temperature was 37·2° C. (99° F.) and pulse rate 100 per minute. He complained of epigastric discomfort, but no real pain, and moved freely in bed. His abdomen was slightly tender in the mid-epigastrium, and more tender in the right iliac fossa. In the epigastrium the muscles were in a state of increased tone, but there was no real rigidity, and further examination revealed no abnormality.

Complete alimentary rest was ordered and the patient was placed in Fowler's position.

At 9 a.m. his condition was unaltered, except that his pulse and temperature were normal. It was discovered that he had been given his breakfast and enjoyed it. An X ray picture of his diaphragmatic region was then taken and a small collection of gas separating the right dome of the diaphragm from the liver was demonstrated (see figure). He obtained his luncheon against orders, and enjoyed it.

At 2 p.m. his abdomen was opened and a small gastric ulcer five centimetres (two inches) from the pylorus, on the lesser curve, was found covered with lymph. On removing the plug of lymph, a perforation about three millimetres in diameter was revealed. No free gas or fluid escaped on opening the peritoneum and there was no sign of peritoneal involvement. The ulcer was infolded with two layers of number 0 catgut and a small piece of omentum sewn over the suture line. Convalescence was uneventful, and the patient was discharged on January 16, 1930.

Comment.

There is usually little difficulty in the diagnosis of perforated ulcers, but there is a definite group of cases in which diagnosis cannot be made with certainty, and in cases of this type the radiological demonstration of free gas in the peritoneal cavity would clinch the diagnosis.

Probably there is no such thing as a leaking ulcer. Perforation is sudden and complete, and three conditions may follow: (i) In the typical case the peritoneal cavity is flooded with gastric contents. (ii) Sometimes the extravasation is localized by adhesions or by protective omentum and lymph, and a local abscess results. (iii) In a few cases, probably because the ulcer perforates when the stomach is practically empty, there is no general peritonitis and no abscess formation. In these cases the ulcer is rapidly plugged with lymph and healing may ensue without operation. It is in Group (iii) that radiological examination is particularly valuable for diagnosis. In spite of the fact that the ulcer of some patients in Group (iii) may heal without operation, they should all be operated on as soon as the diagnosis is certain, in case the lymph seal should lift, with further extravasation of contents.

Singer and Vaughan(1) recently suggested that with more accurate diagnosis we may find these mild perforations or "*formes frustes*" to occur even more frequently than the classical perforation.

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(1) Harry A. Singer and Roger T. Vaughan: *Surgery, Gynecology and Obstetrics*, Volume L, January, 1930, page 10.

Reviews.

PRACTICAL BACTERIOLOGY.

FROM a reading of R. F. Hunwicke's "The Essentials of Bacteriological Technique",[1] it is not easy to determine for whom the book is specially written, for, though within its self-imposed limitations the little work is accurate, there is little in it not already known to experienced workers, and it is difficult to see how a beginner could be trusted to turn out reliable laboratory reports by relying on the book alone. Though the information he would have might be sound, it would be too meagre. We feel the beginner should learn his work under an experienced master in a big laboratory, where books of reference are also big and plentiful. He could undoubtedly take home Hunwicke's book for night study for a couple of weeks to see what might be termed a list of things he is going to learn and then learn them from the big books and the old masters. An ambitious junior can safely be told that this book is accurate, but he would not be long in finding out that it needs supplementing. For instance, there is a lot more to be known in a practical way about filters and their care, about microscopes and their full yield of usefulness. And when the author discusses Gram's stain, giving, incidentally, a good enough modification, he states baldly, "young cultures should be used". Not only so, but any worker reporting on the Gram reaction of an old culture should be dismissed. For young workers under instruction dogmatism should be relentless, not bloodless.

The best chapters in the book are on milk, foods and water, though Wilson's recent work on typhoid bacilli in water is omitted. At the same time the whole work has the merit, pointed out in the introduction, that a multiplicity of methods, some of perhaps doubtful value, is avoided. As a guide to the bacteriology of foodstuffs the book is well worth consultation and this section is decidedly a sound elementary guide to the beginner. Serious omissions in the last three chapters are: the intracutaneous method of animal inoculation, especially in connexion with the diagnosis of diphtheria, and the Martin-Chick method of disinfectant testing.

CORRIGENDUM.

IN the issue of April 23, 1932, there appeared a review of "Ronald Ross, Discoverer and Creator", by R. L. Mégroz. By an oversight the name of the publishers, Messrs. George Allen and Unwin, Limited, London, was omitted from the footnote.

[1] "The Essentials of Bacteriological Technique", by R. F. Hunwicke, B.Sc., A.I.C., with an introduction by W. G. Savage; 1931. London: Williams and Norgate. Demy 8vo., pp. 108, with illustrations. Price: 6s. 6d. net.

The Medical Journal of Australia

SATURDAY, APRIL 30, 1932.

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THIS JOURNAL AND ADVERTISEMENTS.

The functions of the Press are many. Newspapers serve for the dissemination of news. They are a medium for the discussion of political motives and for the gloomy portrayal of a country that is continually going to the dogs. They reflect the efforts of publicists to draw to their own persons the attention of lesser lights or of the non-luminant among the people. They record the social gossip of the bridge table, and incidentally (quite innocently, of course) describe the latest modes adopted by the wives of certain leading medical practitioners with, perhaps, portraits of their quite ordinary offspring. Newspapers have many other functions, and one of the most important is that of advertisement, legitimate advertisement, of course, and not that of the "oblique" variety.

Advertisements in newspapers are of use to the proprietor of the article advertised and to the reader who learns what claims are made for it. If the claims are reasonable and honestly stated, the advantage to the reader must be considerable. The newspaper proprietor, of course, stands to make much money from advertisement contracts; the many full page advertisements appearing in any of the well known daily papers are ample justification for paraphrasing Shakespeare in the manner of "Bovril": "Sweet are the uses of advertisement." Since all goods sold in the open market are not what their manufacturers believe them to be, and since there are always people who will try to foist worthless goods on an unsuspecting and credulous public under cover of a well set out and specious advertisement, it follows that newspaper proprietors may be the unsuspecting accessories to what is in fact a fraud, or may be tempted by monetary gain to advertise substances which they know to be worthless or dangerous. If thoughtful people care to investigate the matter, they will find that most of the newspapers in Australia are subjected to a careful censorship in regard to advertisements. This may be partly, as some might suggest, because honesty pays, but it is largely the result of the high ethical standing maintained by newspaper editors.

If it is important for proprietors of daily newspapers to exercise care in the acceptance of advertisements, it is much more important for those in charge of medical journals to show discrimination. The Medical Journal of Australia, in addition to being a medium for the publication of scientific articles, is a newspaper; but it is a medical newspaper, it is read almost entirely by medical practitioners, and its advertisements have to do with articles likely to be of use to its readers in the treatment of disease and in the conduct of their practices. From time to time it has been found wise to draw attention to the advertisement pages of this journal and to the method of deciding for the acceptance or rejection of a proffered contract. It has been found wise for two reasons, first, because each reader does not realize that every effort is made to exclude unreliable products, and secondly, because advertisers or potential advertisers do not understand the reason for the precautions taken. On previous occasions we have explained that when a product is passed by the committee of *The British Medical Journal* which controls its advertising, the product is accepted by this journal without question; the same holds good for preparations accepted by the Council of Chemistry and Pharmacy of the American Medical Association. When a preparation is manufactured in Australia, an inquiry is made into the process of manufacture, the premises may

be inspected, an analysis of the produce and/or a clinical investigation is carried out. In these circumstances the acceptance of a product by this journal is evidence of our belief in the claims made for it. This is better than any "write-up" that might be given, and is the reason for our refusal to comment editorially on any product appearing in the advertisement columns. If the readers of this journal will bear in mind the care taken in selection of advertisements, they will with the more confidence use the products about which they read.

Current Comment.

PERITONEAL ADHESIONS.

ADHESIONS of the peritoneum may be advantageous or they may be the cause of disaster and death. Many a patient owes his life to the facility with which adhesions are formed in the presence of an acute inflammatory process involving the peritoneum. On the other hand, many a patient has been subjected to repeated operations, has suffered discomfort and illness and has even died on account of adhesions resulting from either inflammation or trauma. Adhesions due to inflammation or trauma often present a difficult surgical problem. Their cure more often than not lies in their prevention—if surgeons handle the peritoneal tissues with adequate care, adhesions do not form. Of course, under certain circumstances the formation of adhesions is inevitable. Much experimental work has been done in this subject, and many expedients have been adopted in the treatment of adhesions. A. Ochsner and E. Garside have carried out experiments with digestive ferments in dogs and rabbits.[1] They point out that adhesions due to peritoneal trauma are usually temporary and generally become absorbed after the inflammatory process has subsided. This is the result of the proteolytic enzyme which is liberated from polymorphonuclear leucocytes as they are destroyed. The experiments of Ochsner and Garside were carried out with trypsin, an animal enzyme and papain, a vegetable enzyme. They performed abdominal operations on the animals with the object of giving rise to adhesions. When the animal recovered from the operation, it was again operated on; adhesions were divided and the enzyme was placed in the peritoneal cavity. When adhesions were divided, they reformed in 100% of cases. If saline solution was added to the peritoneal cavity following division of the adhesions, few, if any, adhesions reformed in 13·3%. If trypsin or papain solutions were added, few, if any, adhesions fromed in 42·2% and 90·8% respectively. Ochsner and Garside have used trypsin and papain solutions in the treatment of fourteen patients. They point out that the period of observation is too short to enable them to draw any definite conclusions. In one instance in which the patient was operated on several times for ileus due to adhesions, trypsin was used; the adhesions recurred. The other patients have remained free from symptoms from six months to five years. In this short account of the work of Ochsner and Garside no mention can be made of the details of their experiments, of the preparation of the solutions of enzyme, of its sterilization or of the concentrations in which they find it can be used with safety. For this information readers must consult the original communication. It is intended merely to draw attention to this work, to point out that it has a logical foundation and that there are occasions on which such a technique might be used with advantage. At the same time many of the adhesions found in an abdomen are the result of rough handling of tissues or of faulty technique.

[1] *Surgery, Gynecology and Obstetrics*, February 15, 1932.

ACUTE ENDOCARDITIS IN CHILDHOOD.

J. L. MORSE has made a critical analysis of attacks of acute endocarditis in one hundred children seen during the first attack ten to thirty years before his article was written.[1] Thirty-six died; three were cardiac invalids and sixty-one were alive and "well". The cardiac condition of six of these was unknown; eighteen had slightly damaged hearts with signs of valvular disease, and thirty-seven had normal hearts. Physicians have stated that children never recover from rheumatic heart disease and pathologists have said that the Aschoff bodies, considered pathognomonic of the condition, never disappear. Accordingly cure is impossible, and at best the disease is only quiescent. Morse observes that he is not sure that he knows what "rheumatism" is, nor does he know whether or not the heart lesions of "rheumatism" are due to allergy. Nor is he certain that it can always be determined whether or not the valvular lesions are vegetative; whether different kinds of sore throat cause different kinds of endocarditis, or whether different strains of streptococci invariably cause different lesions. Morse employs the term "acute endocarditis" not because he considers the condition thus designated to be the only lesion, but because it is the most constant. The term "subacute bacterial endocarditis" was not in use when some of the patients were seen. Possibly a better term would be "acute valvulitis". When the lesion is located mainly in the valves, the heart is but little enlarged, the sounds are strong and the rate only moderately increased. Murmurs constitute the most prominent sign. Morse does not consider it possible to differentiate consistently between vegetative and non-vegetative endocarditis during life. Embolic lesions, however, showed that many cases of endocarditis must have been vegetative. Myocardial implication was demonstrated by increase

[1] *American Journal of Diseases of Children*, October, 1931.

in the size of the heart, with weakening of the heart sounds and considerable increase in the frequency of the heart rate. Pericardial involvement was shown by friction sounds and evidence of fluid in the pericardial sac.

The cases analysed were consecutive and none was included unless the attack was the first one and acute. The time elapsing since the onset of the condition in the patients still living and with normal hearts averaged seventeen and eight-tenths years (varying from ten and a half to twenty-six and a half years). The interval in the cases in which the patients are alive with no endocardial symptoms, but with evidence of chronic valvular lesions, averages seventeen and seven-tenths years (varying from eleven to twenty-five years). The number of complete recoveries is large and the number of deaths relatively small. The age at the time of onset of the disease and the interval elapsing between the onset and the time when the patients were first seen, had no apparent influence on the prognosis. Rheumatism alone or in association with chorea or tonsillitis was the apparent cause in 57% of all the cases of acute endocarditis. If it be held that chorea is itself a rheumatic phenomenon, the percentage rises to 61. In 22% the cause was tonsillitis or other upper respiratory infection. Rheumatism alone or together with chorea or tonsillitis was the cause: in 67% of the fatal cases and of the cases of cardiac invalidity; in 66% of cases in which the patients were alive and "well", but with slightly damaged hearts; in 67% of those alive and "well", but whose cardiac condition was unknown; and in 42% of those in which the patients were living and well, with normal hearts. In only 12% of the fatal cases was the causation ascribed to tonsillitis or other upper respiratory disorder. These same factors were responsible in 23% of the cases in which the patients were alive and "well", with slightly damaged hearts, and in 36% of those living, well and with normal hearts. The prognosis as to life and complete recovery is more serious when, in children, cardiac disorder develops in the course of rheumatism than in tonsillitis or other upper respiratory disorder. These observations, however, are not valid if tonsillitis and nasopharyngitis be deemed themselves to be rheumatic. Investigation of the cases suggests that prognosis is not greatly improved when the patient is seen early. Apparently cardiac murmurs, other than mitral systolic, when observed early, indicate a more serious implication of the heart. Only one of the patients living and "well" has a mitral diastolic murmur. The degree of cardiac enlargement is of value in prognosis, but definite evidence of myocarditis or pericarditis is of greater value. There are many exceptions to such rule. Generally, myocarditis and pericarditis are unfavourable indications. In a large number of fatal cases death occurred early.

Seemingly there are two types of acute endocarditis in childhood, one virulent and rapid in course and the other relatively benign, with good prospect of recovery. The rheumatic children did not all continue to have attacks of rheumatism, chorea or sore throat. However, 40% of the children living and "well", with slightly damaged hearts, had repeated attacks, as compared with 11% of those living with normal hearts. Recurrences of rheumatic infections were not so common as is generally believed, and recovery may occur even when the patient has repeated attacks. When complete recovery does not occur, mitral lesions are not necessarily progressive and deforming, as is generally taught.

The most important factor in treatment is rest, and the degree of care received by the patient is definitely related to the prognosis; the better the care, the better the prognosis. The high percentage of those who died within the first four months who had poor care, was significant. It may have been that infection was virulent in these cases; but lack of care may have made the infection virulent. Some died who had the best care, and others recovered completely with poor care. But, on the whole, the care received by the patients vastly influenced the ultimate issue.

Unfortunately Morse makes no reference to the use of salicylates in rheumatic endocarditis, and it remains a vexed question whether the salicylates are curative or merely palliative in acute rheumatism, or whether even in large doses they prevent relapses. Bacteriology has advanced greatly since Morse's observations began. Many organisms may be associated with acute endocarditis. Streptococci are the most abundant, including *Streptococcus viridans* and *mutans*. In *endocarditis lenta* there have been found staphylococci, pneumococci, *Micrococcus catarrhalis*, *Bacillus influenzæ* and gonococci. These may be only secondary invaders and not causative. *Streptococcus viridans* has been found in the blood in arthritis, rheumatic fever, terminal septicæmia and sinus infections. Subacute bacterial (or infective) endocarditis is rare in childhood and may not be associated with any rheumatic history. It is attended with a high leucocyte count and may often accompany dental sepsis. Frequently it seems to originate or be aggravated by tooth extractions. In many cases of subacute bacterial endocarditis symptoms are referable to the respiratory system, and original diagnoses have been chronic lung infections, empyema, bronchiectasis and subphrenic abscess. Possibly some of the many small infarctions affect the bronchial arteries. Subacute bacterial endocarditis may be symptomless; a case is on record in which an apparently healthy athletic woman had to desist from a game of tennis and died in half an hour. In this instance autopsy revealed coronary embolism with vegetations on the aortic valves. Gram-positive cocci were recovered. *Endocarditis lenta*, which is not yet accurately defined, may display a definite previous history of rheumatism. On the other hand, many cases of acute rheumatism give no evidence of endocarditis. Auricular fibrillation is said to be against the diagnosis of subacute bacterial endocarditis.

Abstracts from Current Medical Literature.

THERAPEUTICS.

"Avertin" and Gas Anæsthesia.

C. Schroeder (*Deutsche Medizinische Wochenschrift*, August 28, 1931) discusses the value of a combination of "Avertin" and "Narcylen" anæsthesia in gynæcological operations. "Avertin" alone is liable to be associated with lowering of the blood pressure and respiratory difficulties. "Narcylen", on the other hand, increases blood pressure, has no effect on respirations, and is an excellent anti-shock agent. In combination they make an ideal anæsthetic. He uses a minimal dose of "Avertin", 0·08 gramme per kilogram body weight, except for very anæmic or fat patients, when the dose is reduced to 0·06 gramme. "Narcylen" anæsthesia is commenced before the skin incision is made and given in a mixture of 40% to 50% "Narcylen" to 60% to 30% oxygen. As the operation progresses, the concentration is reduced to 35% to 40%, and less when the peritoneum is closed. He does not use "Avertin" for Cæsarean section because of the effect on the infant; asphyxia was noted in 21% of his obstetric cases. The details of 100 cases are given and a mortality of 3% is recorded. Two deaths occurred in patients with advanced ovarian carcinoma; the third was due to bronchopneumonia.

Therapeutics and Toxicology of the Sulphocyanates.

J. C. Healy (*The New England Journal of Medicine*, September 17, 1931) reviews the literature on the therapeutics and toxicology of the sulphocyanates and describes severe toxic symptoms which occurred in four patients treated with potassium sulphocyanate. The minimum lethal dose has been stated as fifteen grammes. The commonly accepted dose is 0·3 gramme (five grains) thrice daily for the first week, twice daily the second and once daily the third week. Weakness and dermatitis and nausea and vomiting are indications to discontinue the drug. In all four patients described by the author the peak systolic blood pressure was over 200 millimetres of mercury. Potassium sulphocyanate was prescribed in the commonly accepted dosage. In a male patient, aged fifty-six, with some evidence of arteriosclerosis, the systolic pressure dropped thirty millimetres after three days' treatment, and he became disoriented, developed hallucinations of sight and hearing, and was at times maniacal. The sulphocyanate was omitted, but he required morphine and scopolamine to control the excitement. He became normal after three days, the blood pressure having regained its original peak. A coloured woman, aged sixty, the subject of arteriosclerosis and diabetes, but with normal findings on chemical examination of the blood, had a gradual decline of blood pressure under treatment with sulphocyanate. She became, however, weak and drowsy, and after ingesting 10·8 grammes (180 grains) became comatose, the systolic blood pressure being then 102, and areas of pigmentation had appeared in the mouth and on the flexor surfaces of the elbows. She recovered on cessation of sulphocyanate treatment and on the administration of epinephrine in doses of 1·0 cubic centimetre of one in 1,000 solution for four weeks. The pigmentation continued to deepen for three weeks. Her systolic blood pressure rose to 192. Two female patients, aged sixty-seven and sixty-three respectively, showed a fall of 50 millimetres in the systolic blood pressure after ten days' treatment with sulphocyanate, and developed extreme weakness. In spite of the withdrawal of the drug and attempted stimulation with epinephrine and caffeine hypodermically, the blood pressure continued to fall, and they both died in coma in less than three weeks after stopping the treatment. The author considers that "hypertension as a disease is a generalized condition of the organism which requires increased arterial tonus to maintain function, and that any drug treatment which has the power to reduce this tension to the average normal, leads to disaster". He concludes from the literature, from the cases described and from experiments on rabbits (in which he recovered sulphocyanate, administered transduodenally, from the suprarenal glands) that sulphocyanates in doses large enough to reduce blood pressure appreciably are toxic. The syndrome of hypoadrenia has been produced clinically and experimentally with what have been described as therapeutic doses. The drug is cumulative under certain conditions. Other symptoms of intoxication, such as gastro-intestinal weakness, rash, dermatitis, psychosis, may in fact be manifestations of hypoadrenia of greater or less degree. The incidence of intoxication increases with the age of the patient.

Strophanthin.

F. Grünbaum (*Medical Journal and Record*, October 7, 1931) praises the results obtained by the intravenous injection of strophanthin in the treatment of chronic heart failure. This method is widely used in Germany. It has fallen into disrepute in some countries owing to the use of excessively large doses. It is emphasized that the dosage employed should not exceed 0·5 milligramme in twenty-four hours, and it is best given in two doses of 0·25 milligramme. Strophanthin is contraindicated when there is evidence of coronary artery disease, and when there is severe chronic nephritis. It should not be given to a patient unless he has been free from digitalis saturation for some days. Where small doses of digitalis only have been employed, strophanthin can be used. Many preparations of strophanthin are improperly standardized, and the author recommends only K-strophanthin-Boehringer. Q-strophanthin, to which "Ouabaine" corresponds, is stronger and more dangerous. It should not be used in doses of 0·5 milligramme. Strophanthin can be given intravenously with benefit in any form of chronic heart failure; it requires to be used for a sufficient length of time, as does digitalis; it has the advantage of being non-cumulative in its effects; it is eliminated in twenty-four hours. When œdema does not subside on strophanthin treatment, a combined treatment with the intravenous administration of "Solyrgan" is indicated.

Cirrhosis of the Liver.

M. Roch and H. Wohlers (*La Presse Médicale*, December 12, 1931) record some statistics concerning 431 cases of cirrhosis of the liver treated in the medical services at Geneva between 1900 and 1930. Only cases verified at autopsy are included. It appears that cirrhosis occurs in a large number of persons in Switzerland; Geneva appears to hold the record for the world. Of the 431 cases 418 were cirrhoses of Laennec; 65% of the patients were males and 35% females; the age at onset was about 53 years, and the age at death 55 or 56 years. Alcohol was the apparent cause in the great majority of instances. No particular kind of alcohol was incriminated; but in a quarter of the cases wine alone was the probable cause. In 35% of these cases there were active tuberculous lesions, mainly in the peritoneum and intestine. Syphilis complicated the diagnosis in only 10% of cases. Digestive troubles have been suggested as a cause of cirrhosis by some authors, but in this series the evidence is not sufficient to decide this point. It is possible that the treatment of certain wines with copper sulphate has an influence in causing cirrhosis. The usual symptoms were observed in this series: ascites, icterus, collateral circulation, epistaxis, hæmatemesis and other hæmorrhages. Digestive troubles were the rule. Chronic nephritis was noted in 15% of cases, and seriously aggravated the malady.

Spinal Anæsthesia.

A. Mayer (*Münchener Medizinische Wochenschrift*, September 18, 1931) discusses various objections which have been raised against spinal anæsthesia. The most important is the question of the primary mortality. In his opinion this is not greater than that following inhalation narcosis. He had only one death in 4,200 cases. Later causes of death, such as post-operative pneumonia, do not apply to the spinal method. Unfortunately, in the presence of endocrine disturbances, severe acute anæmia and marked lowering of the blood pressure, the spinal route is not always safe. A second objection is based on the non-abolition of consciousness during the operation. This can be overcome by preliminary medication, also by plugging the ears and bandaging the eyes.

It is also most essential that the operator should handle the tissues and organs most carefully. The Pitkin method with "Spinocain" has enabled anæsthesia to be prolonged. No remedy has been found to prevent the severe headaches noted in many cases. Regarding obstetric practice, he is enthusiastic about its use in Cæsarean section and contraction ring dystocia.

NEUROLOGY AND PSYCHIATRY.

Chronic Subdural Hæmatoma.

ABRAHAM KAPLAN (*Brain*, January, 1932) discusses chronic subdural hæmatoma and reviews in detail eight cases which he has been able to observe clinically as well as at operation or *post mortem*. Though it is less well known as a clinical syndrome, it would appear that this condition is met with more frequently than that of middle meningeal hæmorrhage. The author prefers the term chronic subdural hæmatoma to that of *pachymeningitis hæmorrhagica interna*, which, he claims, should be restricted on pathological grounds to those instances in which the subdural bleeding has resulted from causes other than trauma, and encountered particularly in aged individuals in mental hospitals, in those who are under the influence of alcohol and in luetics with mental changes. Trauma he considers fundamental to the ætiology of chronic subdural hæmatoma—the condition resulting from the tearing of one or more of the cerebral veins as they enter the superior longitudinal sinus with subsequent bleeding into the subdural space. The diagnosis of chronic subdural hæmatoma is suggested when, following a latent interval after trauma, there appears headache of insidious origin, drowsiness, vomiting and coma. A characteristic symptom is a peculiar alternating lethargy with periods of lucidity and alertness occurring within a period of from twenty-four hours to several days. The frequently noted pyramidal tract signs are misleading, but a fixed and dilated pupil, even in the presence of a homolateral paresis, points to a lesion on the same side. Prognosis is grave; but operation, if undertaken early enough, may be followed by recovery.

Psychological Mechanisms in Paranoia.

To understand paranoia is to understand psychiatry. Its mental mechanisms are fundamenteal to the concepts of psychopathology. There is a paranoid trend in nearly every psychotic manifestation. L. S. London (*Psychoanalytical Review*, October, 1931) describes the analysis of a paranoiac of forty-two who was dominated by delusions of the grandiose type. The delusion, representing a symbolic part of his libidinous urge, is fixed; and, unlike the schizophrenic, he has no regression in the intellectual field. The affect is involved to some meaesure in all psychoses. While in schizophrenia the affect is one of indifference, in paranoia the affect is characterized by an emotional reaction to the mood. Paranoia is in fact a disorder of affect.

"Sulfosin" in Schizophrenia.

LOUIS MINSKI, of the Maudsley Hospital, London (*Journal of Mental Science*, October, 1931) reports upon his treatment of *dementia præcox* by injections of "Sulfosin". Of the twenty patients treated, only two appear to have recovered, the condition of one was much improved, one had a short remission, one became much worse and fifteen (75%) shewed no change. "Sulfosin" is a solution of sulphur in olive oil, and is injected into the buttock of the patient with expectations of a febrile reaction. It was introduced some time ago by Schroeder as a substitute for malaria in the pyrotherapy of neurosyphilis. It apparently causes considerable local pain and induration, which may result in stiffness and limping. There is following each injection nausea, anorexia, sweating and transient pyrexia and loss of weight. Tolerance to the irritant develops with successive doses. The procedure, though painful, appears to be free from serious risk, and physical health is rapidly regained after the treatment is terminated. Some patients exhibited exacerbations of their psychotic symptoms during the febrile period; others remained dull and unchanged. The net curative result seems to be no more encouraging than that obtained by other similar therapeutic agents in days gone by.

Epilepsy and the Venous Sinuses.

GEORGE W. SWIFT (*Journal of Nervous and Mental Disease*, December, 1931) discusses the ætiology of so-called "idiopathic" epilepsy. Serial encephalographic studies of epileptics show that the change is one of accumulation of cerebro-spinal fluid over the frontal cortex, then in the cisterns, followed by dilatation of the ventricles and cerebral atrophy. His paper deals with variations and anomalies in the venous sinus system. He claims that the presence of anomalous sinuses—branching of the transverse sinus, accessory transverse sinus and aneurysmal dilatation of the sinus—and asymmetrical development of the transverse and sigmoid sinuses are common in epilepsy; and that the occipital bone in epileptics is frequently smaller than normal, resulting in a small posterior fossa.

Prognosis in Manic-Depressive Psychoses.

HORATIO M. POLLOCK, Director of the Statistical Bureau, New York State Department of Mental Hygiene (*American Journal of Psychiatry*, November, 1931) throws light upon the manic-depressive psychoses. These psychoses are commonly regarded as periodic and recurrent disturbances in the affective life of the individual which may become sufficiently marked as to require certification. The prognosis in regard to the individual attack has been looked upon as favourable, but the liability to recurrence, it would appear, has hitherto been overemphasized. Pollock's figures show that in more than half the cases analysed there was no recurrence necessitating readmission to a mental hospital. He further found that the frequency of recurrence was approximately the same in both sexes and that patients between the ages of twenty and forty years at the time of first admission had fewer recurrences than patients whose psychoses commenced in earlier or later years. The average duration of the recurrent psychotic episodes increases with advancing years. While it is scarcely necessary to emphasize the fallacy of statistical methods generally, it is noteworthy that so many individuals of cyclothymic temperament should have but one episode, whether manic or depressive in type, of sufficient severity as to need mental hospital treatment.

Metastases in Gliomas of the Brain.

HUGH CAIRNS AND DOROTHY S. RUSSELL (*Brain*, January, 1932), believing that the presence of spinal metastases associated with cerebral gliomata has hitherto been underestimated, have reported eight cases in which a primary glioma was found associated with subarachnoid spinal metastases and in six cases with intracranial metastases also. These writers claim that whereas cerebellar medulloblastomata and gliomata of the retina were formerly recognized as the only types giving rise to spinal subarachnoid metastases, their cases definitely establish that the metastases of any cerebral gliomata may, if unrecognized, lead the neurologist astray. Important evidence of metastatic growth may be gleaned from a lumbar puncture showing fluid of a high protein content and xanthochromia. The cases presented included the less malignant and more highly differentiated types of glioma—astrocytoma and ependymoma—as well as the more primitive and malignant neuro-epithelioma of the retina and *glioblastoma multiforme*.

Sciatic Pain.

MANY maladies of widely different ætiology have the common symptom of sciatic pain, such for instance as diabetic neuritis, spinal cord tumours, *paraplegia dolorosa*, pelvic tumours and *tabes dorsalis*. F. G. Lindemulder (*Journal of Nervous and Mental Disease*, September, 1931) who discusses a number of cases, concludes that vertebral arthritis, trauma and foci of infection are the most commonly known causes of sciatic pain, although there is a group in which the ætiology is unknown. After removing such foci of infection as may be discovered, and where other ætiological factors appear absent, the author recommends treatment by sacral epidural injections of 1% "Procaine" hydrochloride in Ringer's solution. He claims to have afforded considerable relief in 87% of cases.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Darling Building, University of Adelaide, on February 25, 1932, DR. A. BENSON, the President, in the chair.

Eye Changes in General Medicine.

DR. BRIAN MOORE read a paper entitled: "Eye Changes in General Medicine" (see page 599).

DR. H. K. FRY thanked Dr. Moore for his interesting paper. He had noticed that patients with marked degrees of defective vision always exhibited a divergence upwards and outwards of the amblyopic eye, also that if he covered his own eye and suddenly uncovered it during fixation, a momentary double image appeared with the shadowy image on the nasal side of the real image. He asked if it was not a normal occurrence, therefore, for exophoria to result in an eye temporarily or permanently deprived of sight. He also inquired whether there was any recognized association between cyclic vomiting and myopia.

Dr. H. M. Jay congratulated Dr. Moore on his admirable presentation of such a very large subject, and expressed the opinion that the ground had been very thoroughly covered. He would add only one suggestion, and that was that anybody examining an eye and finding apparently a monocular conjunctivitis should beware, because such a condition generally turned out to be something else.

In reply to a question by Dr. Lendon, he thought that perhaps the reason why neurotic patients gave such trouble with their refractive errors was that the very nature of their affection rendered them susceptible to smaller degrees of refractive error, and they obtained correspondingly greater relief from correction. It was his experience that this type of patient gave the most trouble in the ordering of glasses, but provided the most satisfactory result in the end.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Armstrong, Thomas Michael John, M.B., B.S., 1929 (Univ. Sydney), 205, Harris Street, Pyrmont.
Kelly, Ronald William Grant, M.B., 1929 (Univ. Sydney), 21, Glen Street, Bondi.

Medical Societies.

THE MEDICAL WOMEN'S SOCIETY OF NEW SOUTH WALES.

THE annual meeting of the Medical Women's Society of New South Wales was held at the Lyceum Club, 156, Pitt Street, Sydney, on March 21, 1932.

Annual Report and Financial Statement.

The annual report and financial statement for the year 1931 were adopted on the motion of Dr. L. Gullett, seconded by Dr. C. D'Arcy. The report is as follows:

The Committee submits the following report.

During the year the Society has made satisfactory progress.

Membership.

The financial membership stands at 92, 17 new members having been elected during the year.

Meetings.

In accordance with the rules of the Society, four quarterly meetings were held during the year.

An extraordinary general meeting was held in June. At this meeting alterations were made to the existing constitution of the Society.

Addresses and Papers.

At the March meeting Dr. Susie O'Reilly spoke of her experiences when a hospital resident and as a general practitioner.

At the June meeting Dr. E. B. Durie gave an address upon the method of obtaining and the significance of the basal metabolic rate, and Dr. Phyllis Anderson read a paper upon the results of recent biochemical research.

At the September meeting an address was given by Dr. Marie Hamilton upon venereal disease, and Dr. Karen Helms read a paper, illustrated by lantern slides, upon infantile paralysis.

Social Meeting.

The Society held its first dinner in December at the Women's Club. There was an attendance of forty members and ten guests, including the recent graduates and the sixth year women medical students. The function was very successful.

Office-Bearers.

The office-bearers elected for 1932 are as follows:

President: Dr. L. Gullett.
Vice-Presidents: Dr. C. D'Arcy and Dr. M. Little.
Honorary Treasurer: Dr. A. Aitken.
Honorary Secretary: Dr. B. Durie.
Committee Members: Dr. Edelsten-Pope, Dr. Hamilton Heighway, Dr. Sandford-Morgan, Dr. I. Saunders, Dr. Florence Voss and Dr. A. Walker.

Cancer of the Uterus.

Dr. C. D'Arcy addressed the meeting on the treatment of cancer of the uterus and of the *cervix uteri*, with special reference to radium treatment as carried out in Sydney and in leading English and Continental institutes. This address was much appreciated, and on the motion of Dr. L. Gullett a hearty vote of thanks was passed to Dr. D'Arcy.

Congresses.

THE CONGRESS OF THE PAN-PACIFIC SURGICAL ASSOCIATION.

DR. F. J. PINKERTON, Secretary-Treasurer of the Pan-Pacific Surgical Association, has announced that the Second Congress of the Pan-Pacific Surgical Association will be held at Honolulu, Hawaii, in April or May of 1933. At the First Congress, held in 1929, delegates from many countries bordering the Pacific attended. It is hoped that in 1933 every country will be represented. Dr. Pinkerton states that the programme is already taking definite form. Australian surgeons are invited to communicate with Dr. Pinkerton, at Honolulu.

Correspondence.

ACCOMMODATION IN MONOTREMES AND MARSUPIALS.

SIR: The very interesting article by Sir James Barrett on the above subject raises some important considerations in evolution that have not, up to the present, received much attention. The facts discovered by his investigations on the eyes of various animals are doubtless correct, and well deserve further work from others. I cannot claim to possess the intimate knowledge of the comparative anatomy of the eye possessed by Sir James Barrett, but what I am going to have the temerity to do is to suggest a possible explanation of how these things came about.

The theory I am bringing forward is briefly condensed in the following proposition. Let us take the three most

important senses in the order of their importance: (i) Eyesight, (ii) hearing, and (iii) smelling. My theorum is that whenever two out of the three are well developed, the third one, owing to neglect, is inferior, and the organ concerned is not likely to be developed to its best. If we consider the case of a mole living underground and making no use of its eyes, naturally we should expect imperfect ocular development; but I do not wish to use this as an illustration, as it is an extreme case and does not bring out what I want. Let us, on the other hand, compare and contrast the faculties of the domestic dog and man. The dog uses mainly the senses of hearing and smelling; man, those of seeing and hearing. Our sense of smell, through ages of neglect, is so inferior to that of the dog that it hardly exists, and we never use it. Now throw a piece of meat to a dog; he may not see it, but quickly, by its smell, he will discover it. He may not even try to see it, but uses his nose at once. Now I don't mean a dog does not use his eyes. A dog has fairly good eyes, I believe, but they are probably inferior to ours and not to be compared with those of an eagle, but he prefers to use his olfactory organ, as it is more highly efficient. I once saw a blind dog, and what struck me was how little he seemed to be inconvenienced by the loss of his sight; when he was among other dogs, the difference was hardly noticeable. He was not being led around by another dog, nor dependent on the kindness of his fellows as we should be.

The best way to learn the methods of a dog is to take him for a walk. This is what you may notice. Another dog appears in the distance; you will see it long before he does, in fact the two dogs will not discover one another till they have come quite near each other, and they do not take a good look, but at once proceed to sniff each other all other; having discovered all they want to know, they assume a friendly, hostile or indifferent attitude; they walk away or perhaps fight, for no obvious reason that we can see. Notice also the dog particularly examines every corner and prominent post, leaving his addition to what he already finds there; this is his little advertisement, for he is a most persistent self-advertiser. He runs his nose along the pavement, discovering the footsteps of previous dogs and, if he had a language, he could tell you something about every dog that had lately been along the street. We could do nothing like that. When a dog meets you for the first time he gives a few sniffs, and quickly decides whether you are friendly or not. See what he does on the hunting field. I doubt if he hardly sees the fox; but he will follow the scent of its footsteps for miles. Imagine how exceedingly minute this scent must be; yet he detects it. Perhaps it is only minute atoms, which our greatest scientists could not discover. We have no sense to compare with this. The nearest approach to it in the possession of man is the capacity of the Australian black trackers; but in this case they can point out marks and slight disturbances, which can be seen. No one can by any means detect the scent of the fast travelling fox. The man on horseback follows the fox with his eyes alone. I could mention other wonderful things a dog can do with this gift, but will refrain. However, not having this sense ourselves, we do not fully appreciate it like many other wonders of Nature, and pass it over as a matter of course. Just try and imagine what a difference it would make if we had it. It would profoundly modify our civilization. A man might come home from his day's work, take a few sniffs in his home and would know who had been there and everything that had happened, and murder or divorce might result. On the other hand, what a valuable gift to doctors! The X rays would not bear comparison with it. A few sniffs and an accurate diagnosis could be made at once by an experienced specialist. I have known a few men who claimed they could smell certain diseases, such as measles, as soon as they entered the house occupied by a sufferer.

Sir James Barrett mentions the marsupials, most of which I am not acquainted with. But there is one, the echidna, which I do know something about, for at different times I have kept three of these most interesting little creatures, and I would like, if I am not making this too long and tedious, to mention a few things I noticed about its ways. Like the dog, its main senses are those of hearing and smell. I regard it as one of the few surviving members of the antediluvian age, a fast disappearing relic. It is a most harmless little thing and, like most of these animals of a bygone period, noted for its powerful means of defence and utter absence of all weapons of offence, also its very small brain. Anyone who tries to keep one will easily realize what I mean. Let it get into a corner or a bit of soil, and you cannot shift it. It has a long, sensitive snout, and a tongue which it is everlastingly smelling in search of an ants' nest. I discovered the simplest way to keep and feed it is to put it in a fair-sized box of straw with a saucer of bread and milk outside; It quickly discovers the food and licks it up with its long tongue. It apparently uses its eyes very little, but has very acute hearing and is exceedingly shy.

Yours, etc.,

T. G. BECKETT.

Melbourne,
February 7, 1932.

PHARMACISTS AND PATIENTS.

SIR: A short time ago a young woman consulted me for a "sore nose" which she had had for four weeks. She complained of feeling extremely tired and of having had a great deal of nasal discharge, mostly blood-stained. She was subject to sore throats, but did not have one at that time. She had consulted a chemist a few days previously, and had been told that she had hay fever. She was given something to inhale; but it had done no good. A nurse advised her to see a doctor.

On examination I found that she was pale, with reddened nostrils which showed some excoriation, temperature 99·6° F., pulse 132. The frontal mirror revealed membrane on the septum and inferior turbinates. Little wonder that she was exhausted and had such a rapid pulse! I gave her 20,000 units of diphtheria antitoxin intravenously. This, with absolute rest in bed and gentle nasal douching, led to such a rapid recovery that the patient herself was surprised. The diagnosis was later confirmed bacteriologically.

I have described this case in some detail because it illustrates my point so well. Pharmacists should exercise the utmost care and discrimination when their customers discuss their ailments with them. The great majority of our pharmaceutical friends are men of sound judgement who realize their limitations; but I venture to suggest that there are some who take too much upon themselves and encroach upon the province of the physician. Is there not much truth in the old adage, "The cobbler should stick to his last"? According to Pope, a little learning is a dangerous thing. We readily agree when we see the errors which result from superficial knowledge. Even those of us who drink deeply by doing many years of hospital work make frequent mistakes. As Hippocrates said long ago: "Experience is often deceptive and a correct judgement is hard to reach."

I know that the chemist is not entirely to blame. To an increasing degree in these days of financial depression the public are inclined to consult the chemist first in the hope that half a guinea will be saved. Secondly, the enterprising manufacturing chemists flood the market with all manner of alleged cures, and it is a temptation to the retail chemist to sell them whenever he can. Pharmacists often see disease in its earliest stages, when the correct treatment would be of most avail. Why not include in the pharmacy course a series of lectures on the subject: "When to advise a customer to consult a doctor, either privately or at a public hospital"? The chemist should forgo the possibility of an enhanced reputation resulting from a cure by a luckily chosen nostrum. I know a chemist who is said to be "as good as any doctor". He is a likable fellow, with unbounded confidence. Socrates despaired of a man who could not be brought to see his own ignorance.

Between doctor and chemist complete harmony, goodwill and mutual understanding prevail. Each is loyal to the other, presenting a united front to an ever critical public.

There can, however, be no acquiescence on the part of medical men to diagnosis and treatment by the chemist. No part of the training of the pharmacist fits him to undertake such work, as most members of that honourable profession realize.

Yours, etc.,
V. H. Wallace, M.D., F.R.C.S.E.

Melbourne,
March 28, 1932.

THE WASSERMANN AND KLINE TESTS.

Sir: Dr. Shannon, in his letter published on March 26, states:

> At the last competitive meeting, held at Montevideo, twelve different methods did not succeed in revealing more than 59% of positive reactions in 623 known cases of syphilis.

This statement is very misleading, because that meeting, similar to the previous League of Nations meeting three years ago at Copenhagen, was only competitive between those methods recently introduced. The older and firmly established methods did not come up for discussion at all. Both of those meetings clearly proved that the older methods alone are reliable. No self-respecting serologist would employ any method, for any purpose, if that method was barely 50% reliable.

Your readers may therefore rest assured that the methods of the old warriors still take pride of place and give 100% positives in untreated cases of secondary syphilis, 97% positives in untreated tertiary cases, 98% positives in *tabes dorsalis*, and 100% positives in *dementia paralytica*.

The above figures are Boas's, from the Serum Institute at Copenhagen.

Yours, etc.,
Alfred E. Finckh.

Sydney,
March 30, 1932.

THE HISTORY OF RECORDING THE BLOOD PRESSURE.

Sir: I have read Dr. Holmes à Court's article on the history of recording blood pressure with considerable interest, but there is one improvement that he apparently has not heard of.

About thirty-five years ago, Professor Charles Martin, now Sir Charles Martin, did an enormous amount of work to make a sphygmomanometer for clinical use that was both portable and accurate. Professor Martin found that the cuff of the Riva-Rocci instrument, as you will notice in Figure X of Dr. Holmes à Court's article, was very narrow, and he made a series of experiments with different size cuffs, and eventually found that no instrument was accurate unless it had a cuff of at least five inches. This work, which occupied several months, has apparently been forgotten, and one notices on the market several instruments where the cuff is under five inches.

It was really Sir Charles Martin who introduced to the general practitioner the accurate and portable instrument such as we now use.

I know this for a fact, as I made some of the glass tubes for Professor Martin, and was also the rabbit during some of his experiments.

Yours, etc.,
Mark C. Lidwill.

135, Macquarie Street,
Sydney,
April 5, 1932.

WORKMEN'S CONTRIBUTIONS TO THE LARGE PUBLIC CHARITIES.

Sir: In the daily papers there is an announcement of a scheme whereby the "workers" on the one hand shall contribute (out of their wages) sixpence a week or less to the funds of the great metropolitan hospitals; and they, on the other hand, shall undertake the treatment of such contributors.

This scheme has the approval of the organizers of the Lord Mayor's Fund and the Charities Board, and a committee is to be appointed, or has been appointed, to consider it; the British Medical Association is to be represented on the committee.

This announcement has not provoked any comment or discussion amongst members of the profession; such apathy is deplorable, but I hope that this letter will call forth some discussion on the matter.

The scheme itself is put forward as a likely means of relieving the deplorable condition into which the great public charities have been allowed to drift. It is, of course, a commencement of nationalization of medical services. I have nothing to say adverse to the nationalization of medical services, when the scheme is a proper one, and when the scheme does not divert the funds subscribed by the charitable public to purposes which were never contemplated by the donors.

In my opinion this scheme offends in both particulars, namely, the scheme is not a proper one, and it will still further divert the funds, subscribed for the relief of the "destitute sick and injured", to the relief of those in sufficiently easy circumstances to afford to pay for their medical attendance.

These two statements of opinion need justification, and I will make an effort to do so.

First, "the scheme is not a proper one". Whilst making a beginning of the nationalization of medical services, the scheme makes no provision for the payment of the doctors. Who would be lunatic enough as to suppose that the Police Force, the Post Office, the Savings Bank or other national concern should be run without paying salaries? And yet, under the spurious name of charity that is what is seriously proposed to be foisted on the medical profession. There is plenty of scope for medical charity amongst the destitute sick and injured; and never will the medical profession fail as a body, though an individual may fail here and there.

Second, the scheme proposes to divert still further the funds, subscribed by the charitably disposed for the relief of the destitute sick and hurt, to the comfort and convenience of those able to pay for medical attention. It is notorious that the big metropolitan hospitals are overcrowded; so over-crowded that all cannot be relieved. Who do you suppose is (reluctantly) refused relief? The destitute poor patient or the patient able to afford to pay for medical treatment? Twice out of three times it is the destitute patient who must be turned away, because all vacancies are occupied by the other class of patient. In my opinion this scheme will aggravate this abuse of charity. If you, Mr. Editor, wish for evidence that, even now, the big metropolitan hospitals are largely used by the class of patient who has no right to charitable treatment, I can supply it in abundance.

What have I to offer in place of this scheme, that I would destroy if it lay in my power? My remedy is a simple one, but bitter: let the hospitals be run on business lines; that is to say, not to spend beyond their income. It is hard, but it is the only safe and sensible way; and it is the only honest way.

Yours, etc.,
A. E. Taylor, M.D.

153, Foster Street,
Dandenong,
Victoria,
April 11, 1932.

THE MEDICAL SERVICE OF THE ROYAL AUSTRALIAN NAVY.

Sir: The subject of your leading article in the issue of the journal for April 9 (the supply of medical officers for service in His Majesty's Forces) has been a bone of contention, and a nasty, smelly one at that, for as long, at least, as my own knowledge of the "interior economy" of

the medical and military professions has cognizance. From time to time, under the urgent compulsion of battle, murder and sudden death, it is buried, to be sniffed out again and fought over by the two parties in the quarrel—the "combatant" and the "non-combatant" sides of both services—when peace and peace-time jealousies and snarlings have replaced the mutual cooperation compelled by some supreme national crisis.

In my considered judgement the real crux is not a question of pay. The matter will be settled when, but not until, the medical service is fully recognized for what in modern war it is—a factor *sine qua non* to victory, not merely, or chiefly, a humane appendage to the "real" naval or military bulwarks of national safety. When such time shall come (as come most certainly it will), then our bone will be burned with mutual consent in the incinerator of common service, whose cleansing fires each side will be equally concerned in keeping clear and effective.

Yours, etc.,

A. G. Butler.

Duntroon,
Federal Capital Territory,
April 11, 1932.

TRAUMA AND ORGANIC VISCERAL DISEASE.

Sir: In your issue of November 7 appeared two papers written by me, one of which was entitled "Trauma in Relation to Organic Visceral Disease". In that paper I covered a certain amount of ground, but I mentioned at the close that there were various other conditions coming properly under the title of the paper which could not be discussed at that time. I specially mentioned hernia, both ordinary hernia and internal hernia, as among these.

I claim the liberty to choose my own subjects when I write. Your correspondent Dr. F. S. Stuckey appears to think he has a right to choose my subjects for me, and that when he proposes (November 21) that there should be a discussion on internal hernia it is my humble duty to obey, although it is a subject about which he has specifically refused (January 16) to write a paper himself. Where is Dr. Stuckey's sense of humour?

In my first paper I mentioned, quite appositely to the discussion, that in two Workers' Compensation cases medical witnesses had stated that muscular effort increases peristaltic action. The reference was to an unqualified general statement, standing by itself as a definite categorical proposition and not depending on any connexion with gastric perforation, with internal hernia, or with any pathological condition or special case whatever.

Now, if this is true, it is a new and important discovery in physiology, and I have expressed the opinion that it should have been published, with all the equally important evidence, in a scientific journal, instead of being revealed only to a few laymen in a Workers' Compensation court.

Dr. Stuckey had other opinions. He sat down at once (November 8) and wrote what he thought (see November 21). I had seemed disrespectful. He wished to protest. It was for me either to prove the thesis false or else to abstain from referring to what had been said. Moreover, I was also barred unless I discussed the whole question of the causation of internal hernia.

I did not take this in the humble spirit desired by Dr. Stuckey, and replied in your issue of December 12.

Then, in your issue of January 16, Dr. Stuckey swings his pendulum right across and says that his former letter was "in no sense a defence of the thesis that muscular effort may increase peristaltic action", and says: "Is it still another of those many queries to which we have to answer 'We do not know'?"

Next, in your issue of February 27, I pointed out the obvious fact that Dr. Stuckey had shifted his position, and asked "What can anybody do with a controversialist like that?"

Once more (March 19) comes a letter from Dr. Stuckey, and here back goes the pendulum to its starting point, just where it was in your issue of November 21. Once more is peristalsis made to result from muscular effort, and once more does he assume that it is my place "to adduce evidence to show its falsity".

My reply is the same as when the pendulum was across at the other side: "What can anybody do with a controversialist like that?"

Now again, on April 16, comes another very long letter from the same gentleman. I have read through it, hoping to find some sign of an inclination to tell sceptics about the evidence on which rests the discovery that muscular effort increases peristaltic action. No, it is still at the point where he left it, namely, that when he himself has spoken, the onus is not on him to prove it, but for another to prove it false (*plus*, of course, the loading of a discussion on the causation of internal hernia).

What Dr. Stuckey does do is to jump another's (Dr. Fry's) pitch, without any respect to the other's rights, and to give us his own views of certain problems in dynamics raised in discussion with the other gentleman, views not bounded by mathematics or limited to anatomical or any other facts, but fancy free. In my two papers I went very carefully over the fundamentals, and kept rigidly to plain, mathematically considered deductions. Dr. Stuckey, seeking relief from the apparently unattractive prospect of having to present the evidence proving that muscular effort increases peristaltic action, thinks to create a diversion by adding words and, I am afraid, subtracting disciplined thoughts. What can anybody do with a controversialist like that?

I want to add just a little more. In his very first letter (November 21) Dr. Stuckey made a sinister suggestion as to the honesty of five men (of whom I was one) with whose opinions he did not agree—a suggestion that was not unobserved either by me or by friends who pointed it out to me.

In his last letter he has done something that still further reveals him. In one of the final paragraphs of my letter in your issue of March 5, where I reply to one of Dr. Fry's, I said: "Let me observe that even if there are peristaltic contractions, there is nothing to prevent us, in the present connexion, from considering the abdominal cavity as practically one space." The "present connexion" was with the fact that the only sort of rigidity possessed by the intestinal tract is that developed by the hydrostatic pressure of its contents against the wall of the viscus. This is all sufficiently obvious to the thinking mind. However, that is not the issue I am raising here, but that this positive assertion of mine, made as "in the present connexion", is actually transferred by Dr. Stuckey into another connexion, not the one belonging to it, and there called a "grudging admission" that peristaltic waves have to be taken into consideration.

This is Dr. Stuckey.

What can anybody do with a controversialist like that?

Yours, etc.,

C. E. Corlette.

Sydney,
April 16, 1932.

[This correspondence is now closed.—Editor.]

DIATHERMY OF TONSILS.

Sir: My experience in diathermy of tonsils does not agree with the claims of Dr. A. B. K. Watkins in your journal of the ninth instant. I have done over one hundred and fifty cases in the past three years by this method, and I have not seen a case of secondary hæmorrhage, nor have I heard a patient complain of pain during the operation. Occasionally I have inadvertently touched one or the other pillar and this was followed by pain the following day, but this has happened only rarely. As for peritonsillar damage, if this does occur, and I am unaware of it, I have not had one complaint from a patient as to any after-effect or disability, and I keep in touch with my patients twelve months after the operation. As my experience seems to be that of most men who have used diathermy extensively, it would appear that Dr. Watkins is writing from hearsay and without reliable information. He is on much better ground when he

speaks of tonsillar remnants being left behind, but I would remind him that in most operations with the guillotine and in many by the dissection method tonsillar remnants are left and extensive trauma is done to capsule and pillars alike. Every general practitioner knows this, and should Dr. Watkins doubt it, let him reexamine cases in any public hospital twelve months after operation. Diathermy is an excellent means of disposing of these remnants. Everything considered, I believe it wiser to remove the whole tonsil, even though, as Dr. Kent Hughes points out, one or two applications usually suffice in most cases. In conclusion, let me quote an extract from *The British Medical Journal* of December 12, 1931, page 1095:

> It is surely time that an English translation of Dr. H. Bordier's "*Diathermie et Diathermotherapie*" appeared. This book, honoured by the *Académie des Sciences* and winner of the *Prix* Montyon (physiology), has now attained a sixth edition in the ten years since it was first published—a proof that its value has been widely recognized. As long ago as 1919 Dr. Bordier was using diathermy coagulation in treating tonsillar hypertrophy and infection, and continues to find the results all that he wishes for. There is never any hæmorrhage, the bactericidal effect is enormous, and the scar non-adherent and supple.

Whom are we to believe, Dr. Watkins, who, I understand, has had little or no experience with the method, or this distinguished gentleman with ten years' practical use of the machine. Perhaps Dr. Watkins regards Bordier a wizard also, but this does not explain away his most inaccurate statements.

Yours, etc.,
F. Wilkie Smith.

Rosebery,
New South Wales,
April 12, 1932.

THE ROBERT H. TODD PRIZE IN MEDICAL JURISPRUDENCE.

Additional subscriptions towards the Robert H. Todd Prize in Medical Jurisprudence have been received as follows:

£5 5s.: Dr. C. B. Blackburn, Dr. Sinclair Gillies.

£1 1s.: Dr. H. G. Allen, Dr. K. S. Macarthur Brown, Dr. C. K. Cohen, Dr. A. J. Collins, Dr. Garnet Halloran, Dr. E. S. Littlejohn.

Diary for the Month.

May 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
May 4.—Western Australian Branch, B.M.A.: Council.
May 4.—Victorian Branch, B.M.A.: Branch.
May 5.—South Australian Branch, B.M.A.: Council.
May 6.—Queensland Branch, B.M.A.: Branch.
May 10.—New South Wales Branch, B.M.A.: Ethics Committee.
May 12.—New South Wales Branch, B.M.A.: Clinical Meeting.
May 13.—Queensland Branch, B.M.A.: Council.
May 17.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xiv.

Austin Hospital for Chronic Diseases, Heidelberg, Victoria: Honorary Radiologist.

Launceston Public Hospital, Tasmania: Resident Medical Officer (male).

The Brisbane and South Coast Hospitals Board, Queensland: Registrar.

The Townsville Hospitals' Board, Queensland: Junior Resident Medical Officer.

Wagga Wagga District Hospital, New South Wales: Junior Resident Medical Officer.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
South Australian: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

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